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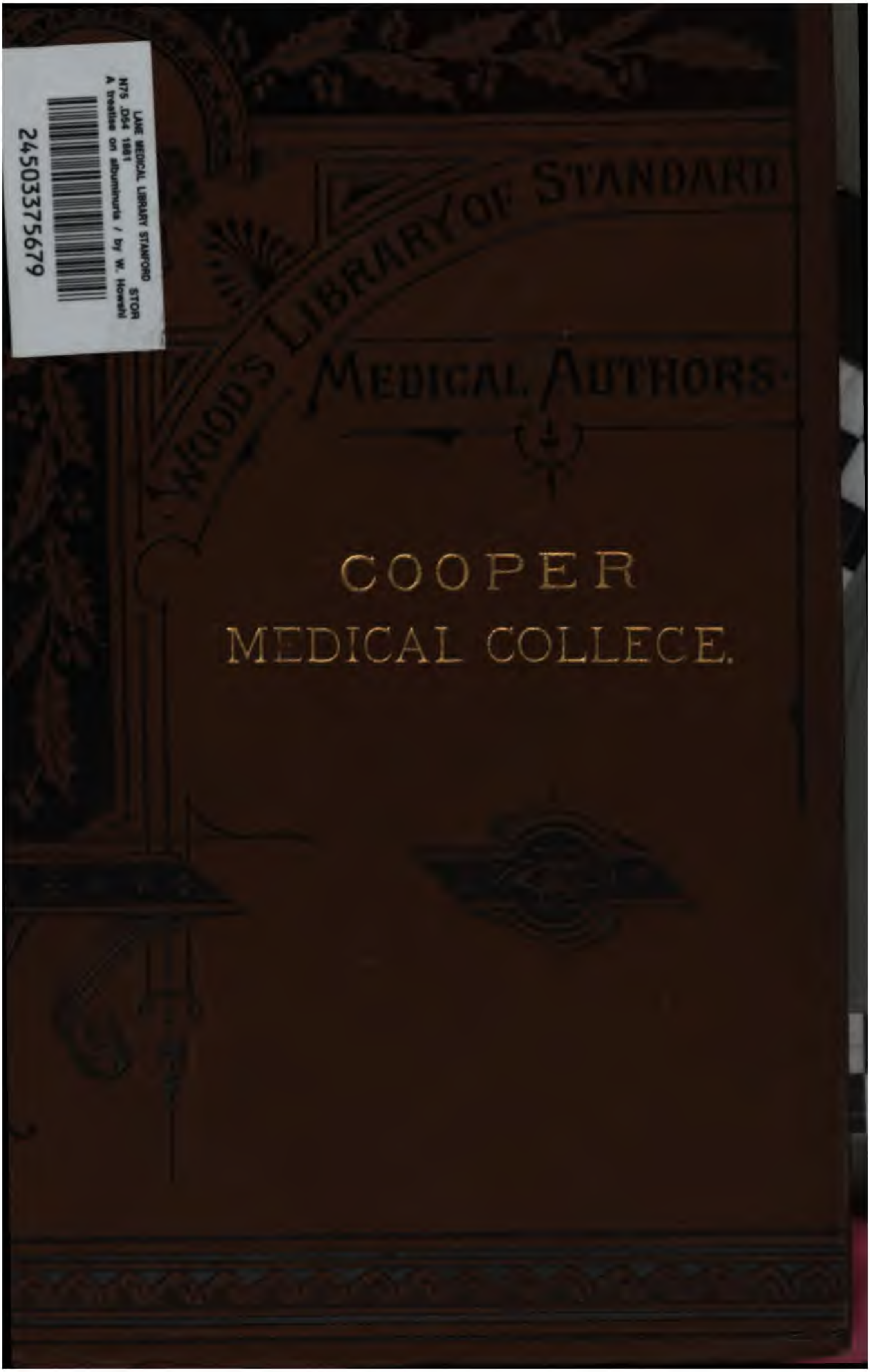

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A TREATISE ON ALBUMINURIA

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BY

W. HOWSHIP DICKINSON, M.D. CANTAB.

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PUBLISHER'S PREFACE.

This volume is one of three written by its distinguished Author upon "Diseases of the Kidneys and Urinary Derangements," and is intended to be a complete treatise upon the subject of which it treats. The Author says of it: "Important questions which have arisen since the first issue have been dealt with in the light of accumulating experience and many illustrations have been added. The leading views formerly expressed remain unaltered. On a point of nomenclature the Author has gladly deferred to the judgment of others, and at the bidding of the Pathological Society has substituted the term *Lardaceous* for Depurative. The suppurative origin of the disorder is, indeed, so evident, and now so widely admitted, that the assertion which the name was intended to convey is superfluous. Improved microscopic methods have carried into further detail the law that renal granulation is but the superficial expression of intertubal disease, and shown the rule to apply even to those cases where granular contraction has ensued upon acute inflammation. These instances, forming a small but well-marked family, once magnified into the general instead of the exceptional origin of the granular kidney, received, in the earlier edition which has been referred to, too little recognition. Their minute anatomy, however, serves but to enforce the distinctions which were urged as essential. When the kidney of tubal inflammation becomes granular it is because the inflammation has become intertubal."

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ALBUMINURIA.

CHAPTER I.

INTRODUCTORY.

THE object of this treatise is to describe those diseases which are made known during life by the presence of Albumen in the urine. These have been classed together under the general term "Bright's Disease," or more recently as Albuminuria. Since the publication of "The Medical Reports"—the work in which Dr. Bright first described the conditions of kidney associated with his name—pathologists have been busy in building upon the foundation he laid.

With the improved means of research which the microscope has given us, and with a rapidly advancing knowledge of animal chemistry, many refinements have been added to the great discovery of Bright.

Many and various subdivisions have been made of the conditions which give rise to dropsy and albuminuria. It would be a weary and unprofitable task to follow pathologists into details of classification, frequently of a highly imaginative character, with which this part of medical literature is burdened. But, while avoiding fanciful distinctions, it will be seen that there are several disorders, different from each other in their cause, their symptoms, and their morbid anatomy, which Dr. Bright associated with albuminous urine.

It is the object of the present volume to remove some of the obscurity and confusion in which the subject has been involved, by establishing such demarcations as are suggested by morbid anatomy, and warranted by clinical observation.

For the purposes of the physician the kidney may be divided into three parts. It consists, first, of a complication of tubes, which, with the malpighian bodies in connection with them, are the sources of its secretion, and are the essential elements of the gland. Secondly, these structures are bound together by a delicate web of sparsely nucleated fibrous tissue, which, in a section prepared so as to show it, has the appearance of a fine uniform network. This pervades the entire bulk of the organ surrounding and supporting every tube and each malpighian body. Thirdly, the gland is abundantly supplied with blood-vessels, which lie with the fibrous tissue between the tubes. Some blood-vessels, indeed, of the smallest size, enter into the construction of the malpighian bodies ; but with this exception the blood-vessels, like the fibrous tissue, are intertubal structures. It is unnecessary to describe with minuteness a structure which will be familiar to every person who is acquainted with the rudiments of physiological anatomy. It is only needful to insist upon the triple constitution of the organ by means of tubes, fibrous tissue, and blood-vessels.

POST-MORTEM STATE OF THE HEALTHY KIDNEY.

Since much of what is advanced in the ensuing pages is based upon alterations found after death in one or other of these components of the organ, it is necessary to ascertain first of all what is its normal condition. It is necessary to know what appearances are presented by the tubes, the intertubal fibrous tissue, and the blood-vessels, in persons who have died of disorders other than renal. It will be necessary to remove from consideration such changes in the kidney as are of post-mortem origin ; and also those variations which depend upon conditions which are common to the whole body, and do not imply any deficiency or peculiarity in the kidneys themselves. The neglect of such precautions has been a fertile source of pathological mistakes.

No detailed description will be needed of an organ so often examined and so well known. The capsule of the kidney is thin and translucent, and holds very loosely to the smooth surface beneath. Upon this surface are no vessels large enough to be singly visible ; but it is uniformly covered with a fine network, the meshes of which are just evident without a glass, and of which each thread consists, not of a single vessel, as might be supposed, but of a multitude of capillaries. On section the well-known brown substance of the cortex separates the cones, and forms a barrier ordinarily about a quarter of an inch in width, between their bases and the capsule. The appearance is liable to much variation within the limits of health, consequent upon the differences in the amount of blood contained ; and a delusive appearance of congestion often rises from staining of the tissues of blood which is unnaturally fluid, as the consequence of febrile disease, or sudden death.

The ordinary appearances of the several renal structures, as seen with the microscope, must be taken into consideration. With regard to the tubes and epithelium in particular it will be needful to be explicit, in consequence of the undue prominence which changes in the epithelial cells have obtained. These cells are sometimes charged with oil globules—a change at one time looked upon as the root of all evil. They have been variously described as "granular," "opaque," "crumbling," "disintegrated," "nebular," "degenerate," etc. It will be shown that each of these conditions may be present independently of renal disease.

With a view of ascertaining what variations of the renal tubes and their contents may be consistent with health, and what must be held to indicate a diseased action, I examined with the microscope the cortex of the kidney in 100 consecutive subjects, in the post-mortem room at St. George's Hospital. The kidneys in 68 cases had the appearance of health, and had belonged to persons in whom there had been no symptom of renal disease. In 32, either in the appearance of the organ or the history of the case, there was some evidence or likelihood of renal change. It must be stated that the microscopic examination in all these cases was made as soon after death as the laws of the hospital permitted.

In the 68 healthy kidneys there were three in which the tubes were not made out. The following table shows their condition in the remaining 65. Many kidneys afforded tubes in several different states.

Table showing the State of the Convoluted Tubes in 65 Healthy Kidneys.

Condition of Tubes.	No. of cases in which present.	No. of cases in which oil globules were found.
Cavity distinct, epithelium in place	9	1
Uniformly filled with epithelium, cells distinguished either by nucleus or outline	44	3
Uniformly filled with amorphous granular matter	13	1
Empty, or containing only a few scattered cells	10	1
Containing fibrinous matter	3	1
Filled with oil globules	3	3
Containing oil globules mixed with other contents	7	7
Tubes distended	6	
Excess of fibrous tissue in cortex	3	
Partial color from solution of iodine	0	

In the 68 cases the state of the cortical epithelium was the following :—

State of Epithelium in 68 Healthy Kidneys.

Typical cells found in	29
Granular contents, but nucleus visible	31
Granular contents, nucleus hidden	14
Opaque, so as to hide nucleus, but not granular	1
Oil globules in cells	25
Colored with bile	1
Yellow or brown color (blood-stained)	12
Small and irregular in shape	1

When epithelial cells are described as "typical," it is meant that the cell is natural in size and shape, the outline and the nucleus well defined, and the contents uniform, transparent and colorless.

From these details it would seem to be rare to find either tubes or epithelium in the state which is generally looked upon as normal. The convoluted tube in its ideal perfection displays a regular arrangement of epithelial cells around a central cavity. The cells themselves are smooth and translucent, with a nucleus clearly visible through a transparent envelope. Such tubes and cells are but seldom exposed to view in the human subject. The disguising power of death is quickly shown upon their delicate structure.

The cells rapidly become granular. They crumble and disintegrate, the outer portion breaking away from the nucleus. They separate from the basement membrane of the tube, and fill the cavity with uniform packing, in which the outlines of cells and nuclei may be recognizable, or may have altogether disappeared in a granular *débris.*

Besides such alterations in texture, the cells often derive a yellowish-brown tint from an unnaturally fluid state of blood. In the twelve cases in which this discoloration of the epithelium was found the blood was thus altered in nine. Blood in this state stains not only the surfaces in contact with it, but the epithelial cells all over the body. It is difficult to say whether the action takes place after death or before.

The renal epithelium sometimes receives a bright yellow color from bile ; this may occur whenever from any cause jaundice is present. Some cells are usually intensely affected, while others completely escape. This change, of course, takes place during life, and is the result of a vicarious secretion on the part of the kidney.

Next to a loss of smoothness and transparency, a deposit of oil between the cell wall and the nucleus is the most frequent change to which the renal epithelium is liable. It is in all cases the result of vital change, not the effect of decomposition after death. After the fatal termination of chronic disease oil is very often present in the renal epithelium, particularly when the disorder has been tuberculous. After acute disease the rule is to find the cells free from oil. In thirty-eight cases in which death had been caused by acute disease or accident, the renal epithelium was found to be fatty in nine. In thirty cases of chronic disease the same change was present in sixteen. Among the cases of chronic disease there were fifteen in which the affection was tubercular ; of these the epithelium was fatty in ten.

The renal epithelium, like the epithelium of other organs, appropriates or parts with oil in obedience to influences which act upon the general structure of the body. The amount of oil contained in the cells of the liver varies with digestion, as well as with the changing phases of disease. After long illness the epithelial cells all over the body are sometimes found to contain visible oil globules. If the disorder had been such as to allow of recovery, there can be little doubt that the cells would have reverted to their former state. Within certain limits, oil globules appear to be very transitory tenants of the epithelial cells, depending for their deposit or removal upon the changing composition of the blood. The presence of oil in the renal cells, even to a large amount, does not necessarily interfere with the action of the gland.

But the change may arise in other circumstances. There may be an especial and local cause to determine the deposit of oil in any particular tissue. The lung, the brain, the kidney, or a portion of muscular fibre, may become charged with oil globules, in consequence of a morbid action limited to the part itself. Extreme fatty change is often found in the superficial fibres of the heart, under the influence of pericarditis, while the deeper fibres escape.

When renal dropsy is brought on by cold, that is when a state of tubal inflammation has been set up in the organ by that cause, it is usual to find the epithelium loaded with oil. The fatty change is the result of the local disturbance. Thus it appears that a deposition of oil in the renal epithelium may coexist with renal disease, as well as with an efficient state of kidney. The alteration may be produced by an inflammatory action in the gland itself, and will then occur in conjunction with other structural changes, and with impaired function. It may also take place in the kidney in common with other organs, in conformity with a general state of system, unaccompanied by any other alteration of structure, or by any sign of renal disease.

So far, then, it has been shown that, without any disturbance in the function of the kidney, there is a great variety in the appearance of the tubes after death, and in the state of the epithelium. The cells may be opaque, cloudy, granular, fatty, variously colored, "crumbling," or broken up, until nothing can be recognized but their nuclei. There is almost as great a variety in the appearances which the tubes may present, though there may have been no renal disorder. If, however, the tubes are obviously increased in width, if they contain fibrinous matter, or if they have lost their epithelial lining, the presence of kidney disease must be inferred.

Besides the tubes and the epithelium which they contain there is a fibrous or fibro-nucleated structure which passes everywhere throughout the gland, supporting, separating, and cementing together the tubes, blood-vessels, and malpighian bodies. About the arteries especially this exists as a decided investure of fibrous tissue. It surrounds the malpighian bodies, not in appreciable bulk, but as a delicate and tenuous capsule. Between the tubes the interstitial material is not definitely fibrous, but exists rather as a translucent and structureless cement in which it is possible to detect elongated nuclei. For its display sections must be specially prepared and somewhat carefully examined. Logwood staining, which displays the nuclei to advantage, shows small elongated bodies of this nature, few and far apart, in a delicate translucent web in the larger interspaces and angular junctures. These are in health inconspicuous. With ordinary methods the tubes appear to be in uniform contact with each other, or with the capsules of the malpighian bodies, and to abut in unbroken array upon the capsule which covers the outside of the gland. The uninterrupted arrangement of tubes along the capsular edge of the section is an important evidence of the absence of intertubal disease. Plate 5, which represents a section which was tinted with carmine, a process not well adapted for the display of nuclei, shows the general appearance and arrangements of the tubes in health.

In certain forms of disease, however, obvious changes take place in these respects. The tubes become widely separated from each other, especially near the surface; the fibrous tissue around the arteries and malpighian bodies becomes conspicuous from its extent, and ultimately large tracts of the organ may become occupied by fibroid or fibro-nucleated tissue to the exclusion of all other structures. In other circumstances the intertubal overgrowth is more uniformly diffused and may even take the shape of a general multiplication of the interstitial nuclei throughout the entire organ.

This preliminary sketch would not be complete without mention of the blood-vessels. Under ordinary circumstances these portions of the gland are not easy to see; it requires that they should be injected to make them distinct. The malpighian bodies, indeed, are conspicuous under the microscope, and if the section happen to include an arterial or venous trunk of considerable size it cannot escape observation; but the vessels of the sizes between the malpighian capillaries and the larger divisions are obscured by the urinary tubes between which they lie. The vessels, undistinguishable in themselves, are not made more evident by the action of any coloring matter, unless it be used as an injection. If the section of a healthy kidney be soaked in a solution of iodine or indigo, the color will be diffused evenly over the whole. Neither of these substances will affect the blood-vessels more than the tubes or fibrous tissue.

It will be seen hereafter that in a certain state of disease the blood-vessels, and other parts of the organ to a less degree, are affected by the reagents which have been mentioned, in a manner which distinguishes them from the structures among which they are placed. The difference depends upon the presence of a material foreign to the normal constitution of the organ, which can be recognized by its peculiar reaction with iodine or indigo.

To sum up the changes which may be relied upon as evidences of disease, they are as follows:—

First.—The tubes may be variously altered by the accumulation of their contents. They may be widened, perhaps irregularly; they may contain a great excess of epithelial growth; they may lose their epithelial lining and become bare; they may become filled with fibrinous matter.

Secondly.—The intertubal fibroid tissue may be increased in two ways, either interruptedly or diffusely. In the first case tangible formations of new fibrous tissue are partially distributed through the gland, chiefly following the arteries, and appearing at certain points on the surface determined by the position of vessels. The intervening structure remains for a time unaffected, while that in the path of the new growth is compressed or destroyed. This change is declared to the naked eye by contraction and superficial granulation. In the second case, that of diffuse intertubal fibrosis, the change belongs to the territory of the capillaries rather than of the arteries. It is evenly spread everywhere between the tubes, where it is rather to be appreciated by hypernucleation than by tangible breadth of new growth. It has its chief importance as a complication and consequence of tubal inflammation, and may exist without any inequality of surface.

Thirdly.—There is a peculiar material, recognized by its "amyloid" reaction with iodine, which appears, if we may make such a distinction, to be rather a chemical product than a vital growth, which is poured out not only into the kidney, but simultaneously into many organs, as the result of a certain state of blood. This substance starts from the blood-vessels and infiltrates the whole organ, giving it the appearance described as waxy or lardaceous. This, and the reaction by which it is identified, is not found save as the result of a very definite disorder, dependent, so far as we know, only upon antecedent disease.

Classification of Albuminuria.

Thus it appears that if we take a rough division of the gland into the three structures which enter into its formation—the secreting tubes with their epithelial lining, the fibrous tissue between them, and the blood-vessels with the blood they contain, we have three loci of disease. Either of these structures may be primarily the seat of morbid change; and the alteration may remain for a long time, or even to the end, confined to the portion of the organ in which it has begun.

Hence we have disease of the tubes, disease of the fibrous tissue between them, and disease of the blood-vessels.

This is not a mere scholastic classification. Kidneys affected in one structure are different from those affected in another, in the appearances they present after death, as well as in the symptoms to which they give rise.

The broad distinctions which separate organs affected in each of these three ways may serve as an introduction to a detailed account of each.

1 First,[1] we have disease of the secreting channels. Like the bronchial tubes and all secreting surfaces, they are liable, under morbid stimuli, to an extravagance of cell growth, which is ordinarily described as inflammation. The disease is an inflammation—if morbidly increased cell growth be inflammation—belonging for the most part to the secreting surface. The tubes, particularly those in the cortex, become loaded with epithelium, which has grown from their walls in extravagant exuberance, or with fibrinous matter, which has exuded from the congested vessels. The tubes are distended, and hence the gland is increased in size, particularly in the cortical part. The large smooth kidney of Bright results. This is pale or congested, as the case may be; at first charged with blood, latterly of a whitish color. The capsule remains thin and loose. The surface retains, at least until the advent of interstitial complications, its gloss and smoothness. In such kidneys the intertubal framework may have entirely escaped; the frequency and completeness of recovery indicates that this exemption is maintained more often than not; but if the inflammation be severe or long-continued a general multiplication of interstitial nuclei may ensue, which will lead to an abnormal growth of fibroid tissue, and possibly at last to granulation and contraction. The disorder may be termed Nephritis, looking at its nature, and as regards its seat, tubal or diffuse.

2 The second form of disease is the contracted granular kidney of Bright—or, as it is termed, granular degeneration. Kidneys so affected are usually smaller than natural, sometimes not half their natural size, the shrinking being chiefly of the cortical part. In the earliest stages of the disorder they may be rather increased in size, but the longer the disease lasts the smaller they become. The great characteristic is a peculiar alteration of surface, which, instead of being smooth, becomes studded with little pale hemispherical granulations. The capsule is thickened, and is more firmly attached than in health. The whole organ is firm and of a fibrous texture.

These alterations are produced by a slow increase, followed by a slow contraction of the fibrous tissue which lies between tubes. The change commences, or at least early appears, at regular intervals upon the surface of the gland, determined by the position of the blood-vessels, producing a depression at each starting-point. The regular arrangement of the depressions produces the superficial granulation which is the great characteristic of the disease. No amount of mere distention of the tubes is able to produce this result. The tubes are of so small a calibre, that, even when distended to their utmost, the equality and evenness of the surface remains unbroken. The increase of the fibrous tissue ultimately takes place throughout the whole organ, surrounding the malpighian bodies by thick capsules, and by its contraction variously narrowing the tubes and cutting them up into cysts. Such tubes as remain open are apt to be affected by the morbid changes which have commenced external to them. The disease is closely analogous to cirrhosis of the liver, and has, indeed, recently and not inaptly been described as cirrhosis of the kidney.

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[1] See a paper by Mr. Simon on Subacute Inflammation of the Kidney. Med.-Chir. Trans., 1847.

These disorders, the tubal and the intertubal, are essentially and primarily renal; they originate in the kidneys, and such other changes as are associated with them are the consequences of the defective action of the gland which these alterations have produced.

The third form of disease, which must be included under the general term, "Bright's disease," differs from them in being the result of a general change, which involves the kidneys in common with other organs. It has its origin in the minute blood-vessels, though not ultimately limited to them. It has been described as waxy, amyloid, or lardaceous.

In reference to its common origin in loss by suppuration—a fact which, I believe, cannot be too much insisted on—I formerly suggested the term "depurative" as fitted to describe the disease in its nature; but there is a large consent in favor of the simply descriptive term lardaceous, in deference to which, and to the recommendation of a committee appointed by the Pathological Society, I shall habitually use it in what follows.

The kidneys, like other glandular organs, become infiltrated with a glassy material, which is poured out of the minute arteries, and spreads itself throughout the whole tissue. The organ first has simply a whitish or anæmic look, it then increases in hardness and in bulk, and assumes, with the prevalence of the new formation, a translucent or "waxy" aspect. The capsule, as the disease goes on, becomes thickened and adherent. After more or less increase of size, shrinking takes place, and the surface becomes uneven, puckered, or undulating. The grand characteristic of the change is in the action of iodine, which imparts to the new deposit a dark brown color, unlike the yellow tint which the healthy parts of the gland receive from the same reagent. This reaction does not take place in any other form of renal disease, and depends upon the chemical condition of the new matter which is poured into the interstices of the organ. It will be shown, hereafter, that the infiltration of this peculiar material occurs especially when the system has been exhausted by a long-continued discharge of pus, though there is reason to believe that other causes, prominently the syphilitic condition, may produce, or largely contribute toward, the same result.

Each of the three forms of disease which have bean sketched will require separate consideration. They affect different periods of life. They arise from totally different causes. They give rise to dissimilar symptoms.

They are unlike each other in their course, in their tendency to recovery, and in the treatment they require.[1]

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[1] This classification was used in the first edition of this work, in 1867. Though similar to one which was indicated by Professor Virchow * in the "Cellular Pathology," I may be permitted to say that it was arrived at independently as the result of researches which had been before the profession since the year 1859. It has been sanctioned by its adoption by many subsequent writers in England and America, and may be said to have met with wide if not general acceptance. The separation of the lardaceous or amyloid form from the other types of renal disease was accomplished by a succession of observers, among whom a leading place must be assigned to the great pathologist of Berlin. These, while not agreeing with each other, or holding the views here put forward as to the nature of the disorder, yet made it apparent that it was isolated and *sui generis*. With regard to the distinction between tubal and intertubal, which forms the basis of the arrangement I have employed, as this has been designated as a German theory, I may at least draw attention to the fact that it is an English theory too. I showed, as I thought, the distinctness of the disorders arising within and between the tubes, in the year 1859, in a Cambridge Thesis, which was published in the Association Journal. The observations on which this paper was founded were made better known in the "Medico-Chirurgical Transactions" of the two following years, in which the changes productive of albuminuria were considered "in relation to their origin in changes occurring in the tubes and in the intertubal structure." What may be the worth of these papers as expressing truths which had not been expressed before, or as expressing truth at all, I leave others so determine. I may at least say that their leading conclusions were due to no previous suggestion. The views in these papers are embodied in this volume.

In justice to a fellow-countrymen, I must state, that in tracing the source of the granular kidney to interstitial fibrosis, rather than to epithelial disintegration, according to the then prevalent view of Dr. George Johnson, I was, as I have recently learned, anticipated as far as the rough notion is concerned—microscopic sections were little understood at the time to which I refer—by Dr. Quain, who suggested the origin of the disease in contractile fibre in a paper in the *Lancet* in the year 1845.

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* "Cellular Pathology," English Translation published in 1860, page 381.

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CHAPTER II.

ON ALBUMEN AND CASTS AS CONTENTS OF THE URINE IN RENAL DISEASE.

ALBUMEN.

WHY should the urine be so generally albuminous when the kidneys are diseased? What are the changes on the part of the kidney which produce, or allow of, this admixture? Is the albumen a new and morbid product, or is it simply that the fluid part of the blood has found its way into the urine?

Such questions must occur to every one when he becomes aware of the symptoms of renal disease, and it may be well to attempt to answer them at this stage of the inquiry, though by means of statements which must find their justification when the diseases are considered in detail.

It is known that the urine may become temporarily albuminous in consequence of an excess of albumen in the blood. The urine of animals has been found to contain albumen when that substance had been injected into the veins or the cellular tissue; and in the human subject the urine has been rendered albuminous in a transient manner by the use of highly albuminous food, as when a large quantity of white of egg has been swallowed after fasting. In this case the result may be in some measure due to the crude or unmodified form in which the egg-albumen finds admission into the blood. It has been said that the same result has followed the rapid absorption of serous fluid from the pleura, a sequence which, to say the least, must be rare. Other morbid conditions occasionally present themselves, especially some exceptional forms of hepatic disease, in which it seems that in consequence of the abeyance of hepatic action an undue amount of albumen reaches the blood without hepatic transformation, only to be cast out by the kidneys. It is not now my purpose to discuss albuminuria save as a symptom of renal disease. Alimentary albuminuria is practically unknown save as the result of experiment; and what may be termed hepatic albuminuria, which appears chiefly to belong to acute atrophy, is, like that disease, of very infrequent occurrence. With this exception, it may be stated as a rule that when the urine contains albumen the kidneys are abnormal either in circulation or in structure. Albumen is a colloid body which transudes with difficulty; its passage through the apparatus of renal dialysis may be generally accepted as an indication that the machine is mechanically imperfect, or is working under unusual pressure.

The urine is albuminous because it is mixed with serum, or at least with its albuminous constituent, which has passed from the blood-vessels into the urinary ducts. There are three conditions, mainly, which are associated with this unnatural leakage. Two relate to the blood-vessels, one to the tubes:—

I. Congestion: undue pressure within the vessels supplying the glandular structures.
II. Lardaceous change in blood and vessels, which is accompanied by transudation of liquor sanguinis.
III. A loss by the secreting tubes of their epithelial lining, in consequence of which they readily yield passage to fluids which otherwise could not traverse them unaltered.

1. Taking these conditions one by one, congestion is shown to be a cause of albuminuria both by the observation of disease and by experiments upon animals. The malpighian bodies are knots of blood-vessels enclosed within the convoluted tubes; an increase of pressure upon the blood within them causes a transudation of serum into the tube; if the pressure be further increased blood-corpuscles also pass out either by migration or rupture. If a ligature be placed upon one of the renal veins these results follow.[1] The urine becomes albuminous or bloody, according to the amount of congestion produced. Such results follow from congestion associated with cardiac obstruction in the human subject. Similar results may follow when the congestion is not of this passive and mechanical nature, but is of the active form produced by a renal irritant. It is probable that, in certain circumstances, the same transudation may be poured into the tubes from the capillaries which lie outside them.

2. In the second place, there is a change in the vessels themselves, which occurs as the essential element in the lardaceous or amyloid disorder, in virtue of which they become unnaturally permeable, or apt to give exit to portions of their contents. The liquor sanguinis, or much that belongs to it, appears to transude in this complaint from the affected vessels, wherever they may happen to be. Such a transudation takes place into the solid tissue of the liver and spleen, from the surface of the stomach and bowels, and in many other situations. The effusion may be so placed as to accumulate within the tissues and interstices of solid organs,

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[1] "On the Circulation of the Blood," by Dr. George Robinson. London, 1857.

or to pass as a discharge from a free surface. The position of the affected vessels determines the result. The malpighian vessels hang loose and free, so that any exudation from them passes into the tubes and mixes with the urine, while the same emanation from the intertubal vessels passes into the interstices between the tubes, and may give rise to new fibroid growth in that situation. When the change happens to affect the malpighian arteries, the escape of liquor sanguinis will, as one of its results, necessarily render the urine albuminous.

3. In the third place, it is found that there is a diseased condition of the renal tubes, which generally coexists with highly albuminous urine. The tubes consist, in health, of a simple membrane, immeasurably thin, upon one side of which are capillary blood-vessels, on the other side epithelial cells. This is the type of all glandular structure. It is probable that the fluid part of the blood can readily permeate the simple walls of capillary and tube, and thus is brought into contact with the epithelial cells. In the epithelial cell lies the power of the gland. The cell acts after its kind upon the fluid presented to it by the basement membrane, and, by the exercise of its function, selects and rearranges the materials which are to form the secretion. When the epithelial cells are removed, the fluid of the blood, having traversed the homogeneous membranes, can pass into the tubes without hinderance or modification.

Such, then, increased determination of blood to, or retention of blood within, the organ, textural changes in the vessel-walls, and loss of the protective epithelium of the tubes, are the immediate circumstances which occasion the admixture of albumen with the urine. Sometimes one may act singly, or any two may co-operate, or all may be conjoined in the same case. How the abnormal conditions arise will appear in the following pages.

CASTS.

What has been said touching albumen applies also to fibrinous casts. Fibrine and albumen equally belong to the liquor sanguinis. When from any of the circumstances which have been described the fluid of the blood enters the urinary tubes, it is only the serous portion which can escape as fluid. The fibrine, if the urine be acid, will solidify as soon as the blood has escaped from its proper channels and reach the urine in a solid form. It appears that in most cases the fibrine is separated in the tubes, which act as moulds to the nascent material, and impress it with their size and shape. Whatever matters the tubes may contain will become imbedded, so that according to the nature of the case the fibrinous cylinder may include epithelial cells in diverse conditions of health or disease, pus globules, blood disks, etc.

Sometimes it is possible, in a carefully prepared section, to trace the fibrinous casts quite up the tube to the position of the malpighian body. In such cases there can be little doubt that they are produced by the consolidation of fibrine which escapes along with albumen from the arterial tuft.

There are other cases in which the casts appear to be derived from the denuded walls of the tubes, or even under the influence of congestion from the walls of tubes which are merely the seat of disturbed epithelial growth.

Occasionally there is evidence of the escape of fibrine in two different parts of the same urinary channel. Casts of large diameter are seen containing within themselves others of smaller size. In such cases the small cast has been formed in the upper, or convoluted part of the tube, probably of fibrine which has escaped from the malpighian vessel. While pursuing its course toward the exit it has reached a wider channel, from the walls of which fibrine is poured out in sufficient quantity to surround and imbed it in a larger cylinder. In cases where I have seen such double casts in the urine I have found after death that the outer investment has evidently been supplied by the straight tubes which have been dilated and bared of epithelium, and have contained large fibrinous plugs. (See plate 3.)

Though as a rule fibrine forms the basis of urinary casts, and often is their only constituent, yet it sometimes happens that cylinders are found in the urine which appear to consist entirely of compacted epithelial cells, or of epithelial cells held together by fibrine so small in amount as to be barely perceptible. Not only do epithelial cells enter into the composition of casts, but, as has been stated, casts may contain anything which traverses the tubes. They therefore give an insight into what is going on in the gland, while by their diameter they show the calibre of the cavity in which they were moulded. If epithelial cells are imbedded, there is a catarrhal or inflammatory state of the tubes, which promotes the growth and detachment of cells. If pus cells are included, the inflammatory or catarrhal state has taken such hold of the tubes that the epithelial cells are replaced by pus globules. Blood globules will show that there has existed enough congestion to rupture, or at least create migration through the malpighian capillaries.

Amorphous granular matter will vary in its indication, according to its origin. It appears that such granular matter is of two kinds, one sort produced by the breaking up of detached epithelial cells, and generally associated with a late stage of tubal inflammation; the other sort belonging to granular degeneration, and apparently consisting of comminuted and altered fibrine.

The diameter of the casts will give indications of a certain kind. If the casts are of small size, not more than $\frac{1}{1000}$ of an inch in thickness, they have come from tubes which retain their epithelial lining. If they are of large size, $\frac{1}{500}$ or thereabouts, they have been moulded in tubes which have lost their epithelial lining. If of larger size still—they may even be nearly double the width last mentioned—they have come from tubes which, in addition to the loss of their cellular lining, have become dilated. Casts of the largest sort may be always regarded as having been formed in the straight tubes, while the smaller varieties may come from the straight or convoluted tubes indifferently.

CHAPTER III.

PATHOLOGY OF TUBAL AND DIFFUSE NEPHRITIS.

It appears that, as regards inflammation of its secreting structure, the kidney bears an analogy with the mucous membranes, especially with such as are bestowed in a tubular form. When the bronchial membrane is inflamed, if the disease be violent enough to destroy life in a short time, we find that the membrane is injected, and that the tubes contain an excess of secretion, which is more or less altered from its natural state. If the patient die later in the disease the excess of vascularity is less evident, and but for the alteration in quantity and character of the fluid which the tubes contain, there is no very legible record of the morbid action which has proved fatal.

In the kidney also the inflammatory change is at first and often altogether strictly limited to the inner surface of the tubes, and the product of the inflammation, as in bronchitis, only a modification of their secretion. There is always, at first at least, an excessive growth of epithelium of which the cells may be nearly natural or simply swollen, or may, as commonly happens in the later stages of the disorder, be fatty. And besides this the tubes may present even from the first, as in every form of inflammation, the results of vascular leakage, blood or fibrine. The narrowness and contortion of the secreting tubes renders it very difficult for the epithelial growth to make its way out, when, as in this disease, it is superabundant. The consequence is that the ducts become packed to distention, and the organ is proportionally increased in size.

It will be convenient to consider the morbid anatomy of the disorder first in its acute, and then in its chronic form. It is not always easy to make the distinction, for the changes are the same in their nature in either case, although the well-marked examples of each sort are sufficiently unlike each other.

In the more acute varieties the inflammatory action, in its early stage, is accompanied by a great increase of blood in the gland, which becomes perhaps more than double its usual weight. I have related[1] an instance in which the capsule was burst, nearly symmetrically in both kidneys, from the extent and suddenness of the tumefaction. But much and early as the kidneys are often swollen from tubal nephritis, such a result is exceptional. The surface remains perfectly smooth, but there is a remarka-

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[1] See work on Diabetes, p. 79.

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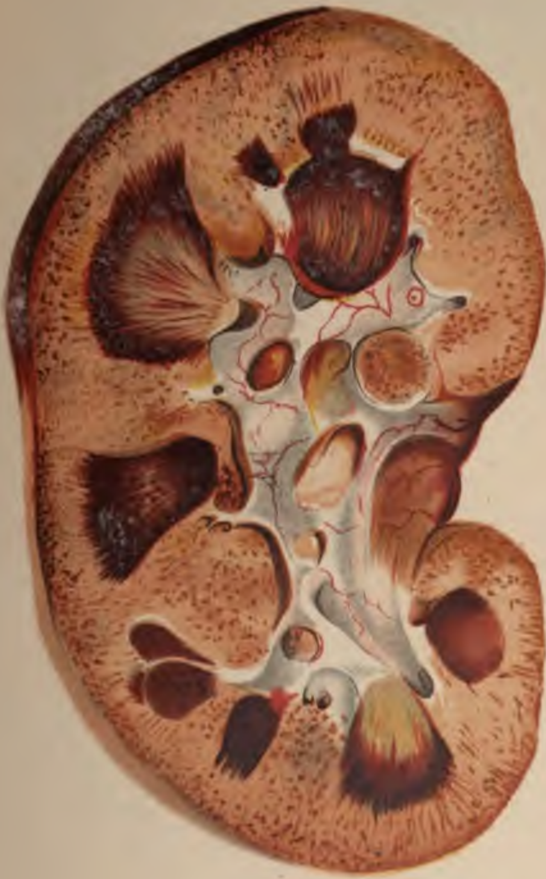
PLATE I., *to face page* 16.

Section of a Kidney in a state of acute Nephritis, the result of Scarlatina. The case is given at length (Vallance, page 70). The kidney is much congested, though that condition is partly hidden by the opaque white matter which occupies the tubes, and causes the increase of size in the cortex. Owing to this congestion, the organ is not so white as it is apt to become later in the disease. The surface of the organ is not shown. It was perfectly smooth.



SCARLATINAL NEPHRITIS [*From case of Vallance Page 97.*]

ble increase of vascularity. The vessels which divide the surface into lobules, and in health are but faintly seen, become intensely injected, sometimes so as to give an almost uniform redness to the surface. The stellate veins which are seen in a later period of the disease are as yet absent. The capsule is loose and thin, as in health. On section the inside presents a red or chocolate color, and drips with blood. The pelvis is injected. Underneath the blood by which the tissue is obscured a light-colored or buff deposit exists, which does not belong to the healthy kidney. This becomes more evident when the surface has been washed. The malpighian bodies stand out as red dots. It may happen that though the disorder be no less acute, the congestion will be less conspicuous than the increase of bulk. The color may be whiter than in health, though the cut surface exudes blood freely, and the whole organ is obviously injected. But the vascularity is masked by the opaque white epithelium which distends the tubes; and the cortex, which is greatly increased, looks as if it consisted of two materials: a red and a buff, coarsely intermingled. The cones are less changed than the cortex, being simply congested. Plate I. represents a kidney in an early stage of nephritis, the result of scarlatina.

Under the microscope[1] the cortical tubes are seen to be stuffed with an opaque brown material, which so long as it remains in the tube does not display any structure, but looks uniformly granular. Spread out on the glass it is seen to consist of cells of epithelium, not changed excepting that they may be stained of a brownish color, beside blood corpuscles and indefinite granular matter; the latter probably resulting from disintegration of the epithelial cells. This condition is most marked in the convoluted tubes, but the straight usually contain more or less of the same material. Besides the cell growth there is usually fibrinous exudation in the tubes. In some cases (Vallance, work on Diabetes, p. 95) this is fatally abundant.

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[1] A few words upon the mode of examining the kidney microscopically may be of service.

The state of the epithelium is best known by scraping the cut surface, and placing a minute portion of the *débris* gathered on the knife, with a drop of water, under a quarter or eighth object-glass.

By this plan tubes are often detached also, but these structures are best displayed in a section. To obtain this a double-bladed knife may be used; but the best way is to expose a small piece of the organ—a cubic half-inch—to the action of a mixture of ice and salt, by which, in about a quarter of an hour, it becomes perfectly hard, so that sections of any degree of tenuity can be cut with a sharp scalpel or razor. Sections made in this manner are adapted to show the effects of reagents, indigo, iodine, etc.

This plan will also show the fibrous tissue, but it is found that the intertubular structures and the attachment of the capsule are seen with more distinctness in the smoother sections which can be made from portions hardened by boiling or by chromic acid.

The best results are obtained by hardening a piece of the organ in chromic acid, and making transparent sections therefrom by a modification of the method introduced by Dr. Lockhart Clarke. A piece of kidney is hardened in a solution of chromic acid (about 1 in 300), or the process of hardening is commenced in chromic acid and completed in spirit. Sections are then tinted with carmine, or, if it be desired to examine the nuclei, with logwood. After saturation with absolute alcohol they can then be made beautifully transparent with oil of cloves, and finally put up in Canada balsam or dammar varnish, in which they will remain as imperishable memorials of the patient from whose body they were obtained. (See Plates V. and IX.)

The beauty and clearness of the sections obtained by this method leave nothing to be desired. The plan is applicable to many other structures.

The malpighian bodies stand out prominently, and their vessel is seen to be distended with blood corpuscles. There are no other changes in the organ. The tubes are everywhere in contact with each other. There is no interstitial effusion, and, excepting the distention of the tubes and blood-vessels, the organ is natural. The disorder—the desquamative nephritis of Johnson—is essentially a renal catarrh. It depends upon a too prolific epithelial growth, not upon any change in the nature of the cells, which, besides swelling, present no tangible departure from their normal state.

The variations in the appearance of the organ depend upon the relative proportions of blood and of epithelium. It seems that the more congestive varieties are produced by exposure to cold, while those in which the epithelial formation is most evident are generally due to scarlatina (see Plate I.). In the latter case the tubes contain little or no blood, but a profusion of natural gland-cells, with some granular material, probably derived from a breaking up of others.

It may be, if the attack is recent and slight, such as often occurs in the course of continued fever or diphtheria, that unless looked at with some care nothing might be noticed but that the cortex is more bulky than usual. The kidney is thick, heavy, and rounded. The cortex often shows a sort of coarse grain, as if a buff-colored formation had been packed in a minute vascular network.

The disease has a natural tendency to recovery. The vascular excitement in which it commences usually depends upon some transient cause, and will subside as it expends itself in secretion, if the circumstances are such that free epithelial growth can take place. This process, however, in the kidney is fraught with peculiar danger, from the narrowness and winding disposition of the ducts. Should the disease prove fatal at an early stage it will probably be in great measure from the plugging of these channels and consequent impermeability of the gland. And, indeed, even to the latest stage of the disorder this condition may contribute to the result. Should the disease fail to come to an early end either by death or recovery, but gradually lapse, as it sometimes will do, into a chronic condition, it will sometimes be found that a change has been superadded which complicates the once simple tubal inflammation by the extension of a similar process to the intertubal tissue. This consists in an universal hypernucleation of the interstitial tissue, discoverable with the microscope, though giving no certain signs by which its presence can be recognized by the naked eye. Woodcuts of this condition are given in work on Diabetes, at pages 28 and 31.

This fibroid hyperplasia may originate in other ways, as will hereafter appear. In the class of cases under consideration it would seem to be a mere superaddition; perhaps the almost necessary result of the long-continued hyperæmia. Its presence can be generally reckoned upon if the disorder have attained several months' duration, and hypertrophy of the heart may be admitted as its clinical indication.

This crosses the boundary which separates the acute from the chronic, and I will now add a few particulars necessary to complete the morbid anatomy of the latter condition.

To look at the organ now in the more chronic condition, or at least at a later stage, it no longer drips with blood when cut open.

In the more serious and confirmed varieties of the complaint there are two conditions which strike the eye—increase of bulk and change of color. The weight is often more than doubled. The secreting structure has a peculiar opaque whiteness, or a pale buff color. When white and ivory-like the epithelium will be found natural; when with a yellow shade it is fatty. The surface is perfectly smooth and glossy, and the capsule readily slips off. The fine network of capillaries which belongs to the surface in health has disappeared, and is replaced by red blotches of vessels disposed in a stellate form, large enough to be followed by the naked eye. On section the pallor of the cortex often contrasts with the cones which retain their normal tint; though sometimes the latter are paler than natural, owing to an extension of the same change as has affected the rest of the organ. The cones are separated from the capsule by an increased thickness of cortex, and are sometimes compressed into the shape of a wheatsheaf. Unless fatty change have taken place the gland is harder than in health.

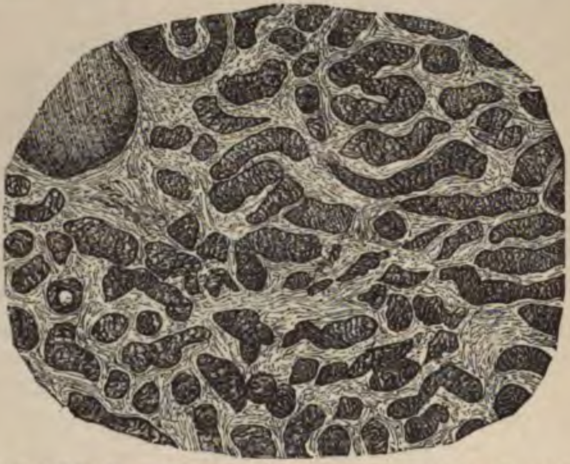
If such a kidney be picked to pieces and looked at with a high power, numbers of distended convoluted tubes will be seen. They contain the epithelial cells either natural in character or fatty, and generally a proportion of them will be found to be broken up into a granular *débris*. Fibrinous exudation is often present.

It was found in an examination of 27 kidneys affected by this disease that the epithelium was free from oil in 10, slightly fatty in 4, generally fatty in 13.

The straight tubes present some variety. Some, like the convoluted, are generally packed with the products of epithelial growth, while others contain transparent fibrine. In the earlier stages this transparent effusion, which has probably come from the malpighian body, is contained inside a natural epithelial lining, while as the disease advances the cells disappear more or less completely, leaving the tube, which now consists only of naked basement membrane, filled with large transparent plugs.

If a section has been cut from a portion of the kidney previously frozen or otherwise hardened it may be seen that all the structures of the organ are unaffected by disease, save only that the tubes are distended. Sometimes all the tubes seem to have suffered alike, so that the whole surface exposed is uniformly covered with dark swollen tubules. Sometimes the distended tubules are more numerous near the surface than in the deeper parts, or may occur in little isolated masses, lying among the natural structure.

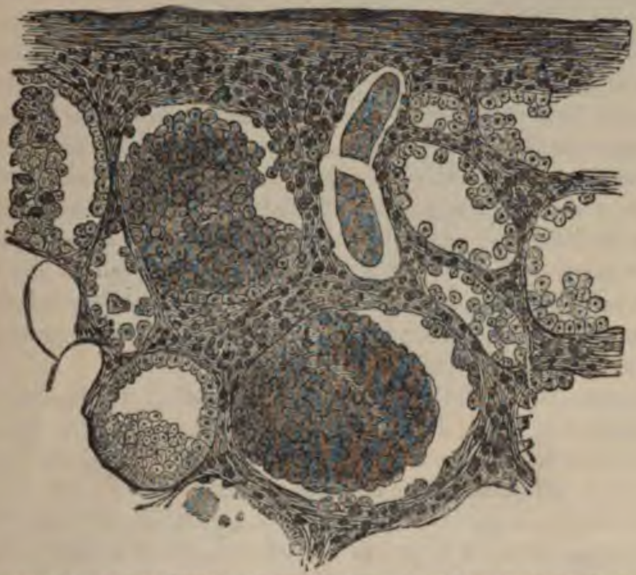
Sometimes the tubes are filled uniformly, as represented in the adjoining woodcut, but perhaps more often, particularly in cases of standing, the obstructions are scattered and partial, some tubes only being plugged, and those only for part of their length. The affected tubes may be stuffed to dark opacity with an epithelial conglomeration which in profile appears solid, but in section often displays a bore which may be either empty or may contain oil globules. Besides such epithelial plugs fibrinous cylinders contribute largely to the obstruction. They are almost always present in the later stages. And sometimes in a very early stage in a rapidly fatal form of the disease they are as abundant as if a general croupous inflammation had pervaded the whole epithelial surface and closed every channel as if with molten glass.



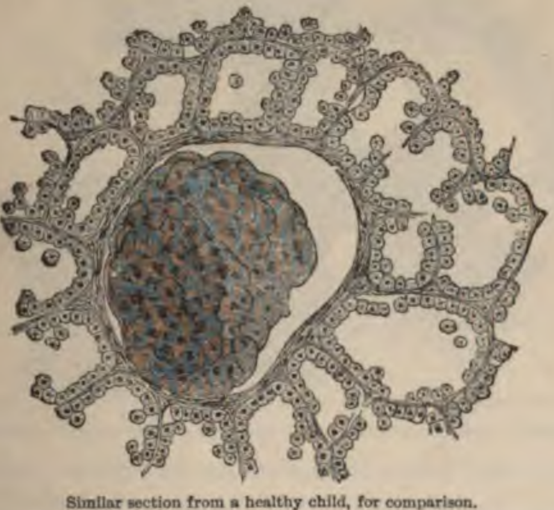
Obstruction of tubes by epithelium, in nephritis of a month's standing; from a child six years old. A malpighian body is seen in the corner.

The irregular or scattered distention which has been spoken of is often marked when the epithelium is fatty; the obstruction is limited to certain families of tubules, which are conspicuous under the microscope by their dark contents, to the naked eye as white specks sprinkled through the tissue. Those of the straight tubes which are affected are marked not by spots, but streaks. A section of a kidney affected as described is shown in Plate II.

Besides such contents as have been mentioned it often happens in recent and congestive cases that a coil of tube will contain blood which has escaped from the malpighian body.



Diffuse nephritis of ten weeks' standing, from a boy of seven.[1] The tubes are variously distended with fibrine and swollen epithelium, and between them is a profuse interstitial nucleation. A malpighian body, surrounded concentrically by nuclei, is seen at the lower part. Almost immediately above it is a tube stretched to nearly the same size.



Similar section from a healthy child, for comparison.

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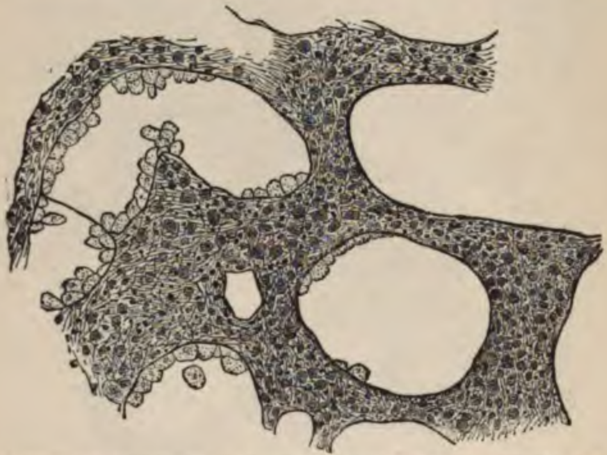
[1] The case to which this woodcut refers is given in work on Diabetes, p. 61.

Such is the condition of simple tubal nephritis; but the kidney offers no exception to the general law that disease to be simple must be recent, and that long-continued inflammatory action, or, in other words, persistent hyperæmia, is apt to give rise to fibroid induration. This in this instance takes the shape of an uniform hypernucleation of the intertubular tissue; attended at first with a scarcely appreciable increase of bulk, but after a time by a nearly even hypertrophy of the interstitial substance and an increase in the bulk of the organ beyond what is due to tubal distention. This change is at first almost inappreciable even with the microscope, and up to a late stage works no such alteration in the appearance of the gland as to make it readily recognizable. But though often undiscovered it is present more often than not in fatal chronic cases; and probably it may be credited in many such with having given the persistence to the disorder which has brought the organ within the range of pathological observation. How often it is absent is seen in the ordinarily absolute restoration of the kidney after tubal inflammation.

It is impossible to say how soon in the course of the disease the additional change may arise; it is certainly absent as a rule in cases which have terminated within the first few weeks; while the majority perhaps of those of which the duration has counted by months show more or less of it. In the instance of the jockey,[1] intertubal cellular formation, though approximating perhaps as much to pus as to fibre, was found within six weeks of the outset. The appearances are represented in the annexed woodcut.[2]

The presence of the intertubal nucleation—or, in other words, of incipient fibrosis—may be suspected when the inflammatory attack is severe and persistent; but there are no early symptoms or peculiarities of cause



Corpuscular formation in interstitial tissue in congestive nephritis, with rupture of capsule—from cortex.

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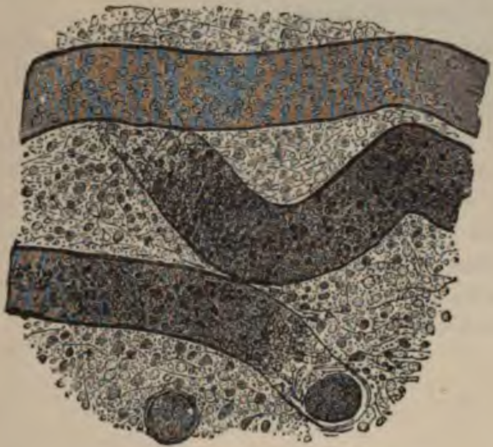
[1] See work on Diabetes, p. 79.

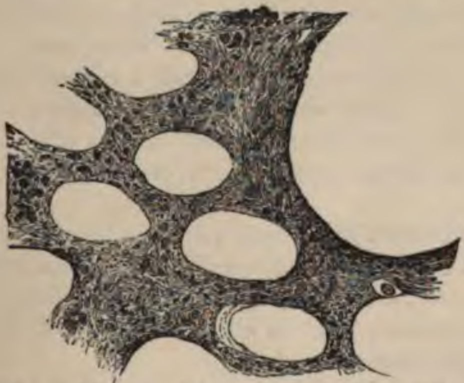
[2] Ibid., p. 30.

or commencement by which the cases to be thus succeeded differ from those which are not. The attack is not to be distinguished from one of uncomplicated tubal inflammation, which, indeed, it probably for a time is. The fibrosis is apparently secondary; its seeds are sown by the inflammation of the glandular elements in which the attack originated.



From cones.[1]

As time goes on, while yet the bulk of the organ is excessive, it gives signs of its contractile tendency in small scattered depressions and superficial retractions. In a small but appreciable minority of cases the fibrotic process survives the catarrhal and pursues its course alone, the contractile



Interstitial nucleation in case of diffuse nephritis, with the large white kidney, of 16 weeks' standing, from a girl of thirteen.

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[1] The case to which these illustrations refer is given in detail in work on Diabetes, p. 79.

superseding every other morbid action. Thus, however different in its clinical origin, the pathological results of this secondary fibrosis may in the end resemble, or even be indistinguishable from, those of fibrosis, beginning independently, as it so often does, in connection with the granular kidney of indeterminate cause and latent origin.

The vast majority of the fatal cases beginning with glandular inflammation terminate with the large white kidney which is smooth or only sparsely dimpled; but now and then in large experience, the disorder having arisen in scarlatina or with acute symptoms from some other recognized cause of renal inflammation, will prove so tenacious and at the same time so tardy, that the end may be postponed for many years, and the kidneys found granulated and contracted. An instance in which this result was found eleven years after scarlatina is related in work on Diabetes, p. 91. Another will be found in the chapter on the condition of the blood-vessels in chronic albuminuria.

Fatty Degeneration.

A fatty condition of the renal epithelium has attracted much attention. As already stated, the epithelium frequently becomes loaded with oil as a consequence of tubal nephritis, especially when that disorder has arisen in consequence of exposure to cold. If the change is extensive and of some standing it gives rise to a striking peculiarity in the appearance of the organ. The outside still remains smooth to the touch, but is closely sprinkled with little white, sharply defined specks, like bits of bran. They have a broken appearance, and their white color forms a contrast with the yellowish cortex, through the whole of which they are interspersed. This appearance is characteristic of a great amount of fatty change in the accumulated epithelium. The specks are aggregations of fat within the tubes. (See Plate II.)

Where the epithelium has become fatty to a less degree it is often difficult to detect the change without microscopic examination. The only difference evident to the naked eye is a somewhat yellowish shade over the cortex, and a coarseness of texture which contrasts with the close fine grain seen where the epithelium is unchanged.

It will be seen hereafter that when the renal disturbance has come on after cold, fatty change is the rule; when after scarlatina, it is the exception. There can be no doubt that the importance of this alteration has been much exaggerated. It is not the primary change, but it is the result of an altered state of nutrition of the cells consequent upon the inflammatory state.

Though rapidly produced it is never seen in the very earliest stage of the disease. It may be found, however, within a few weeks of the

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PLATE II., *to face page* 24.

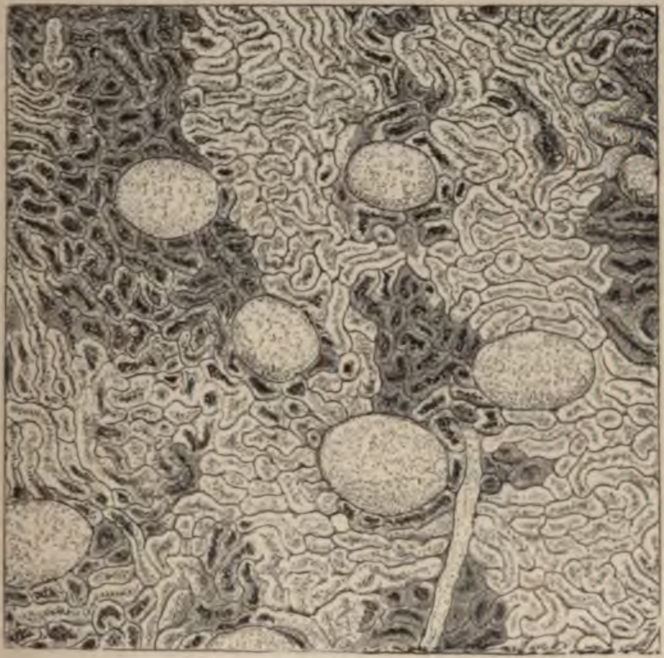
A Section cut from a Kidney affected by Tubal Nephritis, in which Fatty Degeneration of the Epithelium has taken place to a considerable extent. The tubes are not evenly obstructed, but patches of tubes are distended to opacity, while others are clear and translucent. The tubes are all in contact with each other, showing the absence of any intertubular growth.

The Section was made from a portion of kidney which had been boiled.

The organ from which the section was obtained was a beautiful specimen of the speckled kidney described in page 24. The surface was smooth to the touch. The tubes were loaded with fatty epithelium.

The subject of the disease was a boy, who died during an attack of convulsions, having had albuminuria and general dropsy for ten weeks. The cause of the attack was uncertain.

Plate 2.



Magn^{d}. 75 d.

Tubal Nephritis—partial obstruction of tubes.

outset, and may prove fatal within six weeks, though usually more protracted.

The change takes place independently of any similar alteration in other organs simply as the result of a local condition. As in the liver, it by no means indicates a hopeless state of disease. The epithelium, for a time fatty, may recover its natural characters. The only fact I have been able to associate with this state is that it arises in the great majority of cases when the disorder has been traced to cold and exposure. I found that of nine grown persons who had died with the kidney in this state, three were known to have been given to drinking; but, since the condition is found in children, it is hardly possible to assign much importance to this coincidence. It agrees with all our experience in pathology to find oil produced in tissues altered by inflammation.

Considering how much more the kidneys are congested when cold has been the cause of the disorder than when it has followed scarlatina, we may surmise that the alteration is simply due to the greater intensity of inflammatory action in one case than the other.

Such is a general sketch of the pathology of the large mottled kidney, of tubal or diffuse nephritis, with its occasional interstitial complications and consequent ultimate tendency to contraction. It will be easy to add from time to time such particulars as belong to the several varieties of the disease. It will be observed that I have included the fatty and the non-fatty in a common description.

Thus, to place in a few words the results which have been arrived at, they may be thus condensed:

Inflammation arising in the secreting portion of the kidney may take a purely tubal or more general form, the first prevailing in the early stages of the disease, the second in the later. The two are not capable of practical disseverment; they pass into each other by imperceptible grades; they are not to be separated clinically except on the general rule that the longer the disorder has lasted the less simple will be its results; and in their *post-mortem* appearances they are often not to be distinguished save by the microscope, and with that not always easily. It is probable that in most cases the interstitial is merely an extension of the primarily tubal inflammation; evident that in some the inflammatory process is simultaneous throughout the organ.

The following are the more obvious organic results of the various conditions and stages of the process:

1. The blood-dripping chocolate kidney of acute congestive nephritis, usually purely tubal.

2. The large mottled kidney, with absolutely smooth surface and loose capsule, of chronic but usually not very protracted nephritis, as yet purely tubal. Of this the epithelium may be fatty or not according to circumstances.

3. The large white mottled kidney, still smooth or barely dimpled, of nephritis mainly or primarily tubal, but complicated with interstitial nucleation or incipient fibrosis. This is not to be distinguished save with the microscope from the preceding, of which it may constitute either a later stage or a more severe form. As it advances superficial retractions declare its character to the naked eye.

4. The contracted or granulating kidney, not often originating in this manner, but to be recognized as a rare and late result of nephritis primarily tubal.

CHAPTER IV.

CLINICAL HISTORY OF TUBAL AND DIFFUSE NEPHRITIS.

It may be well to consider, first, the conditions under which persons are likely to be attacked; and, secondly, the immediate causes which give rise to the disorder.

SUBJECTS.

It appears that of the adults which are the subjects of it more than twice as many belong to the male as to the female sex.

In childhood this inequality is less. Dr. Miller, in his Treatise on the Kidneys in Scarlatina, has given the details of 66 cases where the disease arose from scarlet fever. His patients, with one or two exceptions, were children; 35 were male, 31 female. I have obtained from my own practice, and that of my colleagues at the Children's Hospital, the notes of 105 cases of the disease. 58 of these occurred in boys, 47 in girls. This includes cases of the disease from all causes.

Dr. Tripe, who has given some comprehensive papers on scarlatinal dropsy,[1] assigns the proportion of males to females as 60 to 39.

I find that out of 16 cases in persons over the age of 16, under my own observation, where the disease, arising from various causes, ended in death, only three were in females.

Hence, it must be inferred that the disease is most common in the male sex at every period of life, and that the inequality is greatest in adult years. Possibly this may be partly due to the more frequent exposure to weather, and the more liberal use of intoxicating drinks in men than in women.

But the fact that the difference exists in children where the habits of the sexes are the same, is enough to prove that the masculine gender is a predisposing cause.

The disease is essentially one of early life, though perhaps no period can claim a total exemption. It is nearly unknown during the first year, rare in the second, afterward common. The frequency with which it happens in children as the consequence of scarlatina, or cold, must have struck every one who is familiar with children's diseases. As to its occurrence later in life, it may be stated to be rare after 40, almost unknown after 50. The later in life the disorder occurs the more congestive its type, as far as regards the affected organ.

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[1] Brit. and For. Med.-Chir. Review, 1854.

Taking the cases at a general hospital, St. George's, I found that of 39 fatal cases which happened under my own observation at that institution, in which the kidneys were examined microscopically, so that there could be no doubt of the nature of the disease, the ages were thus distributed: 11 died during the first 10 years of life, 5 during the second, 7 during the third, 4 during the fourth, 3 during the fifth, while after 50 none occurred. This evidence may be taken as trustworthy, for though the number of cases is small, each has been carefully examined. Much the same results were obtained from the analysis of a much larger number of cases extracted from the hospital records.[1] The prevalence of the disease during the first ten years of life depends chiefly upon the frequency of scarlatina in that period. Thus there are two periods of life which are especially amenable to the disease. The scarlatinal form is most common under 10 years of age; the form which results from cold is especially apt to occur between 20 and 30.

Causes.

Perhaps the only predisposing cause of glandular nephritis which can be clearly traced is residence in a temperate climate. The disease seems to happen with much impartiality in obedience to external circumstances, without reference to any innate or personal proclivity. I have examined with some care the evidence which exists as to any relationship between this disease and the tubercular diathesis. The rarity with which phthisis coexists is such as to lead to the belief that there is no connection whatever between them. Of 29 fatal cases tubercles were found in the body in 4. Regarding the question from another point of view, I examined the details of all the cases of phthisis examined in the *post-mortem* room of St. George's for a term of five years. They amounted to 244. In only 17 were the kidneys described as large, smooth, and mottled; and in the present state of our knowledge we cannot but conclude that most of these were of the waxy or lardaceous variety.

As to gout, which so often accompanies the granular kidney, it is almost unknown as associated with nephritis. In the 106 cases from the *post-mortem* books, gout is mentioned only once, and then in a doubtful manner. Gout, however, leaves so little behind it, that it may easily escape notice in the account of a *post-mortem* examination. I therefore examined the clinical notes of 29 fatal cases of this disease, which had been under my own observation during life, and where particular regard had been had to this question. There was no mention of gout or rheumatic gout in any one.

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[1] See paper in Med.-Chir. Trans., vol. xliv. p. 171.

We may conclude, therefore, that neither tuberculosis nor gout predisposes to tubal nephritis.

As to the immediate or exciting causes these are of peculiar interest in relation to this disorder, since its course appears to be closely regulated by the nature of the cause from which it had sprung. From this circumstance, the clinical history of the disease can scarcely be satisfactory without consideration at the same time of the particular incident which has set it going. It will, perhaps, be well to give first a simple enumeration of the several causes from which the disease springs; then a general sketch of the symptoms which may happen in all cases; and afterward to revert to each particular source of the disorder, describing its mode of action and the consequent course of the symptoms with as much detail as may be needful to make the account complete.

It may be generally stated that this inflammatory affection arises from unnatural stimulation of the kidneys. The blood is charged with material excessive in quantity or unnatural in quality, which these glands take upon themselves to remove. Their own proper elements of secretion are poured upon them in sudden and excessive amount, or matter is thrown upon them which is foreign to their usual habit. As a consequence of overwork, or of work to which they are not adapted, they take on a turbulent and abnormal activity. They become congested, the tubes get choked up with epithelial growth, and the disease is established.

The causes of nephritis may be thus enumerated:

1. Circumstances which throw upon the kidney the work of other glands:—Cold to the body, by checking perspiration; obstructions to the escape of bile; destruction of one kidney, by throwing double work on the other (?).

2. Diseases which develop a material which act as a renal irritant:—Scarlatina, measles, diphtheria, erysipelas, typhus, pneumonia, cholera (?), acute rheumatism (?).

3. Matters taken from without which act as renal irritants:—Turpentine, alcohol, cantharides, arsenic, etc.

SYMPTOMS.

The complaint begins in a definite manner, and can almost always be traced to its cause. The symptom which commonly first attracts notice is œdema. At the same time the urine is darker, and in smaller quantity than usual, and there is probably pain in the loins. The following is a sketch of the disease in its most acute form. A man approaching middle age has been working hard, and while sweating freely, is exposed to cold.

Within a few hours he may become "ill all over," with shivering and headache. Soon his face, particularly about the eyes, becomes puffy, and œdema spreads quickly over the body. He now has a dull pain or feeling of weight in the loins, and the urine has nearly stopped. Perhaps only a few ounces of urine are passed in the twenty-four hours, which is black with blood, and loaded with a dark sediment, which to the eye appears like fine powder. The pulse is hard and full, the skin hot and dry, the tongue coated, the face flushed. There is total loss of appetite, and perhaps vomiting. The older writers often describe such a state of things as acute or inflammatory dropsy, and with truth, though the inflammation is not, as they supposed, diffused over the areolar tissue, but is limited to the kidneys. If the renal mischief be very intense, the urine may be reduced to 2 oz. or 3 oz. a day, and that deeply tinged with blood. This condition cannot last long. If the secretion do not speedily increase, the patient will be poisoned by the retained elements of the urine, become comatose and die. The kidneys, supposing the disease to have attacked a person previously healthy, will be found to be in the state described as belonging to acute nephritis. They will be gorged with blood, greatly increased in bulk, and with an evident deposit showing through the general congestion.

More commonly, however, the patient will not die so early in the disease. The quantity of urine will gradually increase, though still remaining below the natural standard. The dropsy will gain ground, involving the serous cavities, and death will take place in a month or six weeks, with coma preceded or not by epileptiform seizures. Or it may happen that he will be cut off by acute inflammation of the lungs, or of one of the serous membranes.

If the disorder take a favorable turn, the urine will increase in quantity, in some cases even to twice or thrice the natural amount. The dropsy will, at the same time, subside, the albumen at length cease to be perceptible, and the patient will be convalescent, though often left in a very anæmic condition.

It is, however, the exception for tubal nephritis to occur in this rapid and active form. In the majority of cases the disease, though beginning with marked and definite symptoms, and in consequence of a clearly ascertained cause, travels with slower steps. The urine becomes dark and scanty, there is dull pain in the loins, and œdema begins in the face and gradually pervades the whole body. The dropsical effusion varies mostly with the diminution of the urine and the obstruction in the tubes. It extends into the peritoneum, then into the pleuræ, and lastly into the pericardium. Under this more ordinary and slower form of the disorder, anæmia is conspicuous, while febrile symptoms are slight, or altogether absent. Patients thus affected may sometimes be distinguished at a glance from the other inmates of a medical ward. A man under the middle time of life, with a pallid, puffy face, who sits up in a bed, using his arms as supports whereon to elevate his shoulders, may be, without much fear of mistake, put down as laboring under this form of renal disease. And it may be predicted of him, also, that the pleuræ are the seat of serous effusion.

Another form of serous effusion occasionally found in this as in other forms of renal disease, though perhaps less allied in its nature to dropsy than to inflammation, is œdema of the larynx. Cases have been recorded in which the upper part of the tongue, the arytæno-epiglottidœan folds, and the submucous areolar tissue above the vocal cords, have become infiltrated with serum, as a part of general œdema, such as follow scarlatina or exposure to cold. Dr. Gibb mentions such a case in a child of two-and-a-half years old. Dr. Fauvel,[1] in a paper upon albuminuric aphonia, records several instances of the same affection, in one of which tracheotomy was performed with a favorable result. I have met several in my own practice. Laryngeal dyspnœa comes on somewhat suddenly with alteration or loss of voice. On looking into the throat the mucous folds above the epiglottis may be seen to be swollen or puffy, and the epiglottis itself sometimes felt with the finger to be thick and prominent. This, should the case terminate fatally, will be found to be part of general submucous infiltration, which lifts up the membrane chiefly between the epiglottis and the true chords into rounded prominences. The condition is one of much danger, though often amenable to treatment. It not infrequently shortly precedes or directly contributes to the fatal ending. There is usually neither cough nor expectoration. Croupy breathing is its most obvious symptom.

Acute inflammation in less equivocal shapes contributes largely to the mortality of the disease. This is most prone to attack the serous membranes, the pleura and pericardium more often than the peritoneum. The substance of the lungs, also, is sometimes affected, but not so often as the serous investments. Bronchitis is comparatively rare.

With children, inflammation of one or other of the organs of respiration is the most fatal tendency of the disease. Not only are they liable to pleurisy, pneumonia, and bronchitis, but membranous croup sometimes occurs.

Erysipelatous inflammation often attacks the dropsical limbs, though it is to be feared that this is as often the result of injudicious treatment as of the natural tendency of the disease.

Vomiting may happen at any stage, even the earliest; it is often incontrollable. Diarrhœa, on the other hand, occurs but seldom, and then is not a source of danger.

The head symptoms which occur in this more prolonged form of the complaint are usually of a convulsive kind, whereas when the disease is of the acute form first described, coma is apt to set in without any such prelude. The epileptic seizures sometimes come on without any premonitory sign, or they may be preceded by pain in the head, drowsiness, or peculiarity of manner. The convulsive seizures may be repeated in quick succession, and then pass off without any further mischief, or they may give place to a condition of incomplete coma, which is apt to end fatally.

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[1] Dr. Fauvel, Compte-Rendu du Congrès Médico-Chirurgical de Rouen, 1763, p. 33. Gibb on the Throat and Windpipe, 2d edition, p. 282.

The occurrence of any of these symptoms appears to depend on the cooperation of two causes—an anæmic state of the nervous centres, and their impregnation by uræmic poison. *Post-mortem* examination shows the brain, especially in adults, to be pale and watery. Even in cases where much heat of the head has suggested congestion within, it is found after death that the contrary condition exists. It is generally believed that violent convulsive attacks necessarily cause congestion of the brain; but the fallacy of this belief is shown by the facts observed in this disease. The brain is found to be deficient in vascularity, though convulsions have been repeated with violence almost until the moment of dissolution. With children it often appears that the convulsions are associated with some inflammatory state, such as pneumonia, the brain being consequently congested. Continued vomiting appears to act as a forerunner of the convulsive attacks. If a patient, particularly one past childhood, be much exhausted, especially by vomiting, it may generally be predicted that uræmic convulsions are at hand. Much as children are liable to convulsive seizures during the course of other diseases, it appears that with the disorder under consideration they are less liable to be affected by head symptoms than grown people. This may perhaps be attributed to the more rapid course of the complaint in children. A certain time is needed for the production of the general anæmia, and the requisite amount of uræmic infiltration. Adults who are less liable to be cut off by inflammatory attacks, and who at the same time are less likely to make an easy recovery, suffer from what may be considered the ultimate dangers of the disease.

Of 63 cases of tubal nephritis ending in recovery at the Hospital for Sick Children, the subjects being all under 12 years of age, but 5 had convulsive seizures in the course of the complaint.

Taking fatal cases from the same source, I find that in 41 cases, convulsions generally followed by coma occurred in 12, coma without convulsion in 1.

Among persons of the age of 16 and upward the proportion was found to be considerably greater, convulsions or coma occurring in nearly half the fatal cases.[1] It is worth observing, in connection with the state of the brain in these attacks, that they almost always occur with dilated pupils, whereas during congestion of the brain the pupils are generally contracted. When the symptoms take a severe form, the attacks occur in quick succession, sixteen or seventeen sometimes happening within as many hours. The patient between whiles is in a semi-comatose state, possibly with slight stertor, but not absolutely unconscious. The pallor of the countenance will often serve as a ready means of distinguishing head symptoms from this cause.

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[1] See table in work on Diabetes, p. 45.

As consequent upon this disorder when it occurs in its more chronic form, must be mentioned certain affections of the vascular system which, however more common with the granular kidney, are often to be recognized in association with this. Epistaxis occurs, though not frequently. The hæmorrhagic or exudative affection of the retina, which is further described in connection with granular degeneration, is sometimes concurrent with nephritis, especially where the tubal is complicated with the interstitial.

Several instances have come within my observation in which renal disease originating in scarlatina has been more than commonly prolonged, and has at last given rise to the characteristic albuminuric retina.

These obvious secondary affections are but the outward signs of a general modification of the vascular system which occurs as a result of even the simplest forms of tubal nephritis, if only they be sufficiently prolonged. I will not here more than briefly refer to the cardiac and arterial changes which are associated with renal disease, since they are discussed in detail in a subsequent chapter, but I think it beyond doubt, as a matter of observation, that the heart becomes hypertrophied and the arteries thickened from the continuance of simply tubal nephritis, or at least of nephritis in as simple a form as is consistent with its long continuance. If the disorder last long enough to cause secondary arterial changes the kidneys will be found seldom to have escaped at least a microscopic amount of incipient fibrosis or interstitial nucleation. Thus hypertrophy of the heart may be practically recognized in a case of inflammatory albuminuria, as a hint that secondary changes relating not only to the heart but to the kidneys are in progress, and the disease transferring itself to the category of the irremediable. The increase of arterial tension, the hardness of the pulse and the evidence of the sphygmograph, are enough to show that extraordinary resistance is opposed to the circulation even at the outset of the disease, and that this should cause cardiac hypertrophy and arterial change is consistent with views which we may fairly hold with regard to the origin of these alterations.

The table on next page gives an abstract of the symptoms which occurred in a series of 39 fatal cases, of all of which careful notes were kept.

The table has been divided, so as to show how the symptoms differ in childhood and adult life. It seems that œdema is almost an invariable symptom, and that ascites and hydrothorax stand next in frequency. Vomiting is characteristic of the disease rather than diarrhœa. Uræmic poisoning is the great danger in the adult; in childhood inflammation of the respiratory organs. The tendencies of the disease at the two periods of life will be further considered.

Not to pass without notice a branch of clinical observation, which with regard to some disorders is of the first importance, it may be said that the variations of bodily heat in tubal and diffuse nephritis are scarcely characteristic. I have before me a considerable number of temperature charts be-

Table showing Affections consequent upon Tubal or Diffuse Nephritis in Childhood and afterward.

Affection.	Under sixteen, twenty-three cases.	Sixteen and upward, sixteen cases.	Total number, thirty-nine cases.
Hæmaturia[1]	11	7	18
Frequency of micturition	0	4	4
Pain in loins	1	9	10
Œdema	22	16	38
Ascites	11	9	20
Hydrothorax	7	4	11
Fluid in pericardium	1	0	1
Purpura	0	1	1
Epistaxis	0	2	1
Erysipelas or abscess	3	5	8
Uræmic convulsions	5	5	10
Simple coma	0	1	2
Other head symptoms	4	0	4
Pneumonia	9	1	10
Pleurisy	5	8	8
Peritonitis	2	3	5
Pericarditis	0	1	1
Endocarditis	0	0	0
Bronchitis	8	0	8
Coagulation of blood in pulmonary artery	0	2	2
Croup, or diphtheria	4	0	4
Vomiting	4	5	9
Diarrhœa	2	3	5
Gout	0	0	0

longing to these disorders in their different forms and rates of progress. Some betray extreme and irregular elevations, others maintain much the even tenor of health. It is generally manifest that the variations are due not to the renal disease of itself, but to associated and secondary affections, either to the fluctuations of some febrile disorder in which the albuminuria may have originated, or to superaddition of one or other of the many inflammatory affections which are so often consequent upon it. The thermometer in these cases, like the barometer at sea, is chiefly useful in foretelling storms.

When the renal disease has been apparently uncomplicated I have often known the temperature to range for many weeks between 97.5 and 99.5, and sometimes to keep for considerable periods within narrower limits. With an uncomplicated and chronic case the range of temperature may be that of health or possibly somewhat depressed. Perhaps the only elevation we can assign directly to the disease is a rise occasionally appreciable at the outset, especially in that sudden and congestive form of the disorder which succeeds upon cold. I have no thermometric record of a rise at outset of more than a few points above 100°, but that the temperature is sometimes much raised in the circumstances I have mentioned has been occasionally evident to touch, though no accurate observation has been secured.[1]

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[1] Blood evident to naked eye.

The preceding outline chiefly applies to nephritis where it ends fatally, fortunately by far its less frequent termination. From whatever cause it arises, it has a natural tendency to get well, stronger or weaker, according to the age of the patient, his previous habits, and the source of the complaint. Children have a better chance than their seniors. As resulting from febrile disorders, by far the majority of cases will recover under judicious treatment. When from cold, the recoveries are, as will be presently shown, fewer. Even with the most confirmed and seemingly hopeless cases recovery will sometimes take place, and it is not a solitary experience for a patient with this disorder, discharged as incurable, to come back in perfect health long after he had been thought to be dead. Recovery is heralded by an increase in the quantity of urine, which soon comes to surpass its natural measurement, often to a great degree ; at the same time the dropsy is carried off, first from the areolar tissue, then from the serous cavities. I once knew in such circumstances twelve imperial pints, or 240 ounces, to be passed in the twenty-four hours, carrying with it not only the dropsy, but a large excess of urea, the result of previous accumulation. The urine may recover itself soon after the dropsy goes, and the patient speedily return to health ; or in a less favorable case the dropsy and all conspicuous signs of the disease may subside, while the urine continues albuminous perhaps for years. Casts and renal epithelium can generally be detected after the albumen has ceased to be perceptible.

Sudden Attack of Dropsy, with Bloody and Albuminous Urine—Recovery.

The following case, which furnishes an instance of recovery from a severe attack of nephritis, is slightly abridged from a paper in the "Lancet," Nov., 1861, by Dr. Williams of Swansea. The patient was a medical student, twenty-two years of age, in whose fate, as Dr. Williams tells us, he was deeply interested. If it is allowable to hazard a conjecture, we may guess that the patient whose symptoms are portrayed so faithfully is no other than the distinguished physician who tells the tale.

"At the end of the winter session at one of the London hospitals, at which he had been severely working, and while enjoying his ordinary health, this gentleman was seized in the night with bloody urine, and a frequent desire to pass it. The urine was first observed to be dark in color, and much reduced in quantity on rising in the morning. His suspicions were excited as to this symptom, and he went to Dr. Barlow, who tested the urine, and found upon the addition of nitric acid that it was literally converted into one clot of albumen. Dr. Bright, who was consulted the next day, recommended the patient to leave town for the country, holding out no hope of recovery. Dr. Prout, who was next appealed to, said that the case was one of hæmotrophy of the kidneys, the urine being bloody, of high specific gravity, and highly albuminous. Satisfied now that he had become the subject of Bright's disease, the patient resolved to leave for the country. Neither Dr. Bright nor Dr. Prout would hold out any hope of recovery.

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[1] Some valuable information upon the temperature in renal disease will be found in a paper by Dr. E. L. Fox of Bristol. Med. Times, Oct. 1, 1870.

"On the tenth day after the first appearance of the symptoms the patient went into the country. The urine was still scanty, less than three-quarters of a pint in twenty-four hours; the dropsical symptoms every day increased; the urine was as dark as porter, and highly albuminous; the skin was dry and feverish; the breath was growing in tightness and difficulty. In a fortnight further the body had greatly increased in size, while the urine had scarcely at all augmented in quantity. Convinced that he was going to die, greatly alarmed at the swelling and the difficulty of breathing, the patient determined to resort to compound jalap powder and warm baths, after which a change for the better became evident; the skin began to act, and the urine increased in quantity. Thus he continued for three months. The urine was now more abundant, and contained a smaller quantity of albumen and of blood.

"Under the use of citrate of potash and iron, which were prescribed by Dr. Prout, the improvement continued for six months, at the end of which time the dropsy had entirely disappeared, the patient had become pale and thin, the urine had greatly increased in quantity, the micturition was frequent, both during the day and the night. The urine deposited masses of casts, epithelial cells, oil globules, red corpuscles, and granules.

"The albumen continued to diminish; the urine averaged three pints in the twenty-four hours, was of a slight muddy or smoky tint, and never fell below 1012 in sp. gr. The patient was pallid, nervous, and dyspeptic, but was active in mind and body.

"At the end of two years from the first attack the albumen had entirely disappeared. For three or four years afterward there persisted a slowly decreasing amount of renal irritability—that is, micturition frequent at night, and the ordinary secretion was rapid and excessive during the day, if at any time a stimulus were taken. After the urine had ceased to contain albumen the microscope still showed a few casts, cells, and oil globules.

"More than twenty years have elapsed since the patient was downstruck by his attack. He is now [1] in perfect health. The urine has been perfectly healthy for at least fifteen years. He has never experienced the slightest relapse or return of the original symptoms.

"Six years before the beginning of this illness the subject of it had a severe attack of scarlet fever, followed by slight dropsical symptoms."

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[1] That is, when the first edition of this work was published, in 1867.

DURATION OF THE DISEASE.

It may be well to give in this general sketch a few facts as to the duration of the disease in different circumstances. The first six months will, in a large proportion of cases, bring the issue to recovery or death. Most of those who die do so within this period. But to this rule there are striking exceptions. The disorder, originating perhaps in scarlatina, or in some other equally definite cause of renal inflammation, and beginning with a sudden outbreak of dropsy, will change its pace and character and drag its victim wearily to the grave after the manner of the more chronic forms of renal disease. In such cases the interstitial growth, probably at first absent, has crept slowly into the place of the subsiding catarrhal disorder and written its indelible fiat upon the struggling organ. Five years ago I lost sight of a girl, then sixteen years of age, who had been frequently under my observation with albuminuria, the result of scarlatina thirteen years before. An instance is related in which a young woman died with a contracted and granular kidney, the issue of scarlatinal dropsy dating eleven years back; and I might accumulate in no inconsiderable tale examples in which disease of the same character and origin has been protracted for several years. In such it may be stated as a general rule that the kidney will present more or less evidence of contraction in superficial puckering or even granulation, and that its interstitial tissue will be found to have increased. Such cases, in fact, may have come to present pathologically and clinically almost all the characters of the primarily granular kidney. But these, though not few in large experience, are insignificant in their number as compared to those of which the issue is determined within the comparatively brief limits previously mentioned.

Recovery may take place at any time, however late, so long as the tubal disturbance remains uncomplicated; but it is sufficiently clear that the longer the disorder lasts the greater the chance of secondary fibrosis. It is not unknown, however, for the disease to stretch over several years and to eventuate in recovery. In such cases the albuminous state of the urine long outlasts the more visible symptoms.

The time which the disease takes to reach a fatal ending depends much upon age. The younger the patient the more rapid the course to recovery or death. The accompanying table gives the duration of the disease in fatal cases, at the several periods of life, childhood, adolescence, and adult age. It is compiled from the notes of 54 instances in which *post-mortem* examinations were made at St. George's and the Children's Hospital. It will be seen that during childhood the majority terminated during the first month, many within the first week. Few survived the third month, none the fifth. After the age of 20 no deaths took place within a week, few within the first two months. The greater number of cases thus ending do so after the end of the second month, before that of the sixth.

Table showing the Duration of Fifty-four Fatal Cases of Tubal or Diffuse Nephritis, arranged according to the Age of the Patient.

	Under 4 yrs. old.	4 to 10.	10 to 20.	Over 20.	Of all ages.
Dying within 1 week..................	2	4	—	—	6
Between 1st week and end of 1st month	4	10	1	2	17
In 2d month..........................	2	6	2	1	11
" 3d "	—	3	3	4	10
" 4th "	—	—	—	1	1
" 5th "	—	2	—	3	5
" 6th "	—	—	—	3	3
Later................................	—	—	—	1	1

IMMEDIATE CAUSES OF DEATH.

Before concluding this general sketch of the symptoms of nephritis it may be interesting to consider the circumstances to which death is generally due. These vary with age; children and adults not only die, as has been shown, at different periods of the disease, but they die in a different manner.

I have collected the particulars of 50 cases fatal under the age of 16 which were examined *post-mortem*—40 from the Hospital for Sick Children, 10 from St. George's. 28 of these owed their deaths mainly to inflammation of the respiratory organs. This number includes 16 instances of pneumonia; 10 of pleurisy, of which 5 had gone on to empyema; 8 of bronchitis; 6 of croup or diphtheria, the latter possibly due to extraneous causes in the shape of hospital influence; 1 of œdema of the glottis. In many, as is evident from the figures, the inflammation was not limited to one structure. Pneumonia in particular frequently concurred with pleurisy and with croup; and œdema or serous infiltration of the lung, though nowhere appearing as the chief cause of death, contributed to it in many instances.

Twenty-two cases remain to be accounted for. Among these death was caused by uræmic convulsions in 8, by dropsical accumulation in the pleural cavities in 5, by vomiting in 3, by peritonitis in 4, by pericarditis in 1, by sloughing of scrotum in 1.

Of the age of 16 and upward the patients endure the disease much longer and allow it to proceed to what may be considered its legitimate conclusion. They are not cut off by pneumonia, pleurisy, or any other intercurrent affection, but generally live long enough to die of uræmic poisoning, or of one of the direct consequences of dropsy. In these cases, as the disease draws to a close, it is usual to find several secondary affections attacking the patient at the same time; it is therefore difficult to assign the fatal issue to one alone. If the patient have convulsions the chances are that he has had obstinate vomiting. If he have extreme dropsy he is too likely to have erysipelatous inflammation, or abscesses in the cellular tissue as the consequence of acupuncture.

Analyzing 15 cases, fatal in the adult, and selecting the affection to which death appeared to be mainly due, it was found that in 2 only was it produced by inflammation of the organs of respiration, 1 by pneumonia, 1 by pleurisy. In 5 it was due to convulsions or coma, in 4 to dropsy or its effects, as erysipelas or superficial abscess ; in 2 to peritonitis, in 1 to pericarditis, and in 1 to coagulation of blood in the pulmonary artery, the lungs being otherwise diseased.

The Urine in Tubal and Diffuse Nephritis.

The changes which the urine undergoes are variations in quantity—diminution in the early and progressive stages, sometimes great increase during recovery—the addition of albumen and sometimes of the crystalloid constituents of blood, of tube casts, and sometimes of blood in its entirety.

The more scanty the urine, and the greater the proportion of albumen in it, the more intense the disease, a statement which does not hold good with the granular kidney. There is no more promising sign in tubal inflammation than a spontaneous increase in the amount of the urine. At the outbreak of the disease the urine generally, though by no means invariably, contains an amount of blood enough to cause obvious discoloration. When the urine is acid the blood will give a black or smoky tinge ; when alkaline or neutral, a pink or red color. As the disease advances the secretion loses its brightness and amber tint and becomes dull and deficient in coloring matter.

I will revert in brief detail to some of the points which have been touched upon.

It is reduced in quantity, except during convalescence, when it is often greatly increased. When the disease occurs in an acute form the urine may be reduced in quantity to between half an ounce and 2 oz. daily. This is the minimum, and only occurs in cases where the tubes are almost universally stopped up with epithelium or fibrine. In the case of Benjamin Patrick, in which the disease followed exposure to cold,[1] the urine fell to 5 oz. in 24 hours. In that of the jockey mentioned in work on Diabetes, page 79, also consequent upon cold, the urine fell to 5 drachms in the 24 hours : the total passed during the last five days of life being 3½ oz. Vallance, with scarlatinal dropsy,[2] passed on one occasion less than 2 oz. in the 24 hours. The degree of diminution reached in these instances is fortunately rare, since it is a symptom of the worst omen.

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[1] See work on Diabetes, p. 76

[2] Ibid., p. 95.

Diminution to a less extent, the urine being reduced to half or a quarter its natural amount, is of constant occurrence, so long as the disease is stationary or progressing. During the process of recovery the urine is often greatly increased. As the tubes become clear the diuretic action of the retained materials becomes effective, and the secretion may be twice or thrice its normal amount. This spontaneous increase is a favorable prognostic. The scantiness of urine is in direct proportion to the obstruction of the tubes.

I may allude to the instance of a young man who, while recovering from an attack of acute renal dropsy, in which the urine had been much lessened and solid with albumen, began to pass water in increasing quantities until 240 oz. was reached as the maximum for 24 hours. The secretion then slowly lessened, the average for seven days being 167 oz. The specific gravity may be generally stated to be much what it is in health. 1019 may be given as the average during the disease. In the most acute cases, where the urine is very scanty, it may be much above the natural mark ; later in the disease, when the secretion has become more abundant, it is often at 1010 or thereabouts.

When the urine is allowed to stand a copious sediment is thrown down, particularly when the complaint is of recent origin. In the absence of blood the sediment will consist of the contents of the tubes. There will be seen multitudes of cells of renal epithelium, which may be natural or fatty ; and perhaps, especially in advanced cases, a few pus corpuscles may be detected.

Furthermore there will be casts. These may occur in great abundance, sometimes so much so as to form by themselves quite a palpable sediment, They present considerable variety. Some are much more common than others, and some come early and some late in the disorder. They may be thus classified :

Epithelial Casts.—Transparent fibrine, enclosing epithelial cells, or cells packed together into a plug, are found in almost all cases, probably in all, at some time or other. They belong especially to the early periods of the disease.

If the epithelium passes off, not in entire cells but broken up, the casts are *granular* in character, often opaque and coarse. These are as truly epithelial in their structure as those where the cells are seen entire and distinct. If the epithelium of the kidney becomes changed by disease, the contents of the casts will vary accordingly, and display fatty cells or pus, as the case may be. The granular casts generally appear in the more advanced stages of the disease, since it is necessary for their production that the epithelium remain in the tubes long enough to become disintegrated.

Transparent cylinders of fibrine—small, as from a tube duly lined with epithelium ; or large, when from one which has lost its epithelial growth—are often found. These are the "waxy casts" of some writers. When small they are apt to be overlooked from their great transparency. They

PLATE III., *to face page* 40.

Fig. 1.

State of the Tubes in Nephritis. The epithelial lining has generally been removed, while the tubes are occupied by epithelial cells and fibrinous matter. Both the epithelium and the fibrinous matter are in some cases dotted with oil-globules. Both convoluted and straight tubes are seen, the former being the more distended. (See p. 20.)

Fig. 2.

Casts of Tubal Nephritis. All the casts represented were obtained from patients in whom the nature of the disease was placed beyond doubt, either by post-mortem examination or by the fact of recovery having taken place. Most contain epithelial cells; some, granular matter. Some are simple cylinders of fibrin. One bears evidence of having had a double origin, as explained in page 14, a large cast including others in its interior. (See p. 41.)

Plate 3.

Contents of tubes in Tubal Nephritis.

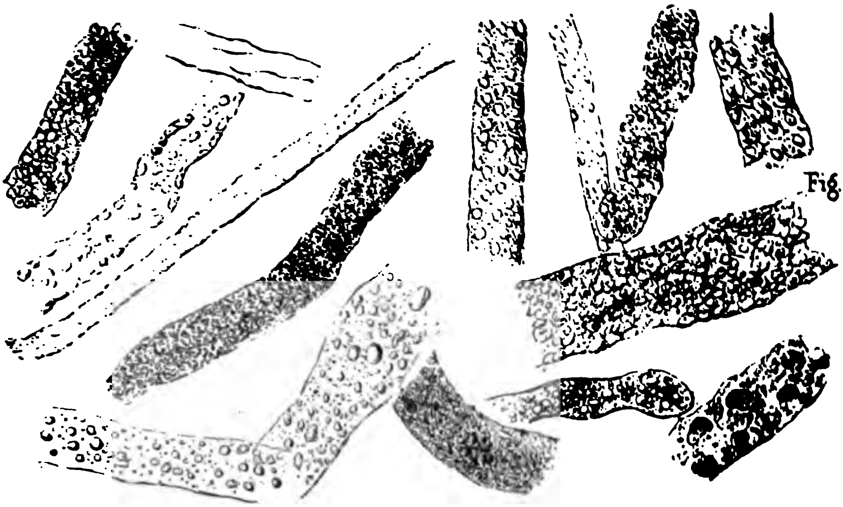

Fig. 1

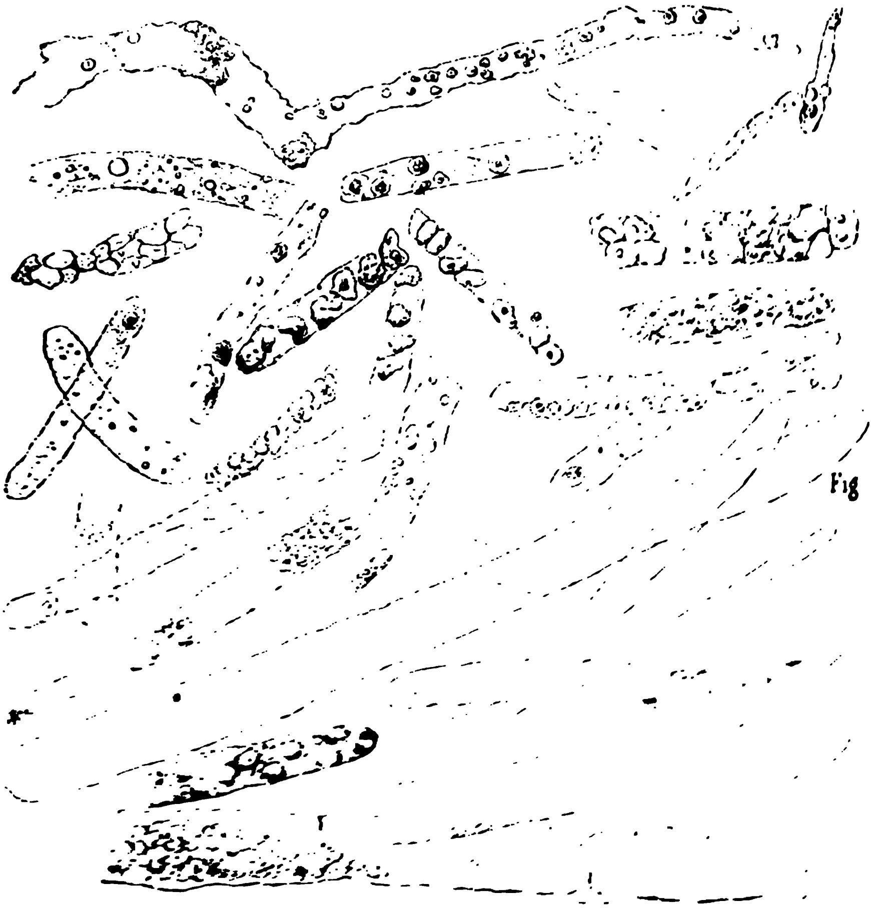

Fig 2

Casts—Tubal Nephritis.

are usually found in this disease, though they are by no means peculiar to it. The small and the large are very distinct in character and in origin. Those of small size may be found, as has been shown, in simple congestion. The large cylinders are definite and distinct; they only occur in the most advanced stages. They sometimes imbed smaller casts in their interior. They come from the straight tubes.

During the presence of hæmaturia the casts may contain blood-corpuscles, or may have a brown color from being tinted by hæmatine.

Hence, it seems that, judging by the casts alone, it would be often impossible to be sure of the nature of the complaint. If, indeed, a quantity of loose renal epithelium were found with many epithelial casts, we might safely venture upon a diagnosis. Casts containing pus-globules could scarcely admit of more than one interpretation, but they are of rare occurrence. It must, of course, be borne in mind, in endeavoring to estimate the condition of the kidney by the microscopic characters of the urine, that it is not uncommon for some amount of tubal nephritis to complicate the course of granular degeneration, or waxy infiltration. In such a case epithelial casts and epithelial cells will be seen in addition to the deposit belonging to those diseases. (See case of Charlotte Carter, related as an instance of lardaceous disease.)

Chemical Changes in the Urine.

The urine is altered by the addition of blood, or one or more of its constituents, and by the diminution of all the natural urinary components.

Taking the superadditions first as the most characteristic, it is not necessary to say more about the presence of blood than has already been advanced. Albumen, invariably present, is abundant. The commencement of the disorder is generally marked by a decided coagulum of albumen in the urine—a fact not observed with other forms of renal disease. It affords, by its quantity, a generally correct measure of the severity of the attack. A diminution in the amount of albumen is as favorable a sign as an increase in the quantity of water. The amount of albumen sometimes discharged is so great as to amount to an appreciable if not an injurious drain upon the system. The quantity may be stated to vary from a trace up to 35 grammes (more than an ounce) of dry albumen in the 24 hours.

In the instance of a man under my own care at St. George's, where the urine was remarkable for the solidity with which it coagulated, I found that the albumen in 24 hours came to 21.9 grammes, or three-fourths of an ounce. In another, where the urine was less intensely albuminous, but had increased with convalescence to 118 ounces, the albumen reached the maximum of 32½ grammes. But perhaps little is to be gained by an exact estimate of the quantity. The increase or diminution from day to day may be sufficiently ascertained by the rough method of boiling, acidulating with nitric acid, and measuring with the eye the bulk of the coagulum. This varies from a mere cloudiness, such as is noticed during convalescence, to a bulky clot, which in some cases is large enough to convert the whole quantity of fluid operated upon to a gelatinous or solid mass.

The recognition, by means of guaiacum, of the crystalloids of blood is to be mentioned as among the early signs of the disease, one which may, indeed, precede the appearance of albumen in the urine. A state of urine, indeed, in which the crystalloids only of the blood are discoverable has been described as a prealbuminuric stage of albuminuria; and it would seem that at least in some cases the sapphire blue imparted to the urine by its admixture in a test-tube with tincture of guaiacum and ozonic ether declares the presence of those elements of the blood before the less fluent albumen has been able to traverse the coats of the vessels. This reaction, according to Mr. Mahomed,[1] accompanies a general increase of arterial tension which precedes the local renal inflammation, and constitutes a stage of the disease in which it is amenable to prevention, however obstinately at a later epoch it may resist curative measures.

Passing to the normal components of urine, the variations of water need no further notice than will be found in the previous statements with regard to the quantity of the secretion.[2]

As to urea, so long as the disease is stationary or on the increase, this product is diminished. The amount generally varies with the amount of water. When the latter suffers the extreme of reduction the urea is usually reduced to a very small quantity. In a case of fatal scarlatinal dropsy reported by Rosenstein, the urea fell as low as 1.4 grammes in the 24 hours. The patient was a girl 15 years of age. In the case of Vallance the urea fell to .72 grammes in the 24 hours, probably not a twentieth of the normal amount. This extreme diminution is a symptom of the worst import, and

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[1] See paper on the Etiology of Bright's Disease and the Prealbuminuric Stage, by Mr. Mahomed, Med.-Chir. Trans., 1874.

[2] As a standard of comparison, it may be well to give the mean of the urinary constituents in the healthy male adult. The following amounts for 24 hours are taken from Dr. Parkes' valuable work on the urine:

Quality, 1501 c.c., or 52½ oz.
Sp. gr., 1020.

Urea	33.0	grammes,	or 512 grains.
Uric acid	0.555	"	or 8. 5 "
Phosphoric acid	3.164	"	or 48 "
Sulphuric acid	2.012	"	or 31 "
Chlorine	8.21	"	or 126 "
Chloride of sodium	13.6	"	or 210 "
Soda	11.09	"	or 171 "

Potash, varying from 1.7 to 7.6 grammes, or from 26 grains to 107 grains.

is usually followed by nervous disturbance—generally convulsions. In most cases, however, the diminution is more moderate. In the case of a man who died of the disease after 25 days' illness, the symptoms having been brought on by cold, the urea varied from 15.7 to 8.2 grammes in the 24 hours—from half to a quarter the proper amount.[1]

In a case reported by Becquerel, in which the patient ultimately recovered, the urea amounted to 11.64 grammes in 24 hours.

In three successful cases of scarlatinal dropsy under my own care I found that in general terms the urea fell to somewhat more than half its proper quantity. In a child of 4 years old the minimum was 11.25, against 22.25, which it became after recovery. In a child 7 years old the minimum was 16.5, against 29.22, which it became after recovery. In the third case, a child of 9 years old, the minimum was 15.24, the amount after recovery being 28.0.

In these cases, as is usual during convalescence, the amount of urea was no doubt somewhat above the normal rate.

And as an example of the increase of this excrementitious substance under the increase of urine which sometimes accompanies recovery, I may mention that a man already alluded to passed while losing his dropsy 240 ounces of urine and 47 grammes of urea in 24 hours. The daily average for 5 days amounted to 42 grammes.

Though it is not uncommon in this disorder to find a deposition of uric acid, in a crystalline form, or as urate of soda, this appears to result more often from scantiness of the urine than excess of the acid. This is the case particularly with children, and in the earlier stages of the disease.

But sometimes in the later stages, when the urine is no longer scanty, a considerable deposit of uric acid may be noticed, such as to indicate a positive increase in its excretion, as if the kidneys with returning function were removing an accumulation. The general rule, however, is that the uric acid, like the other urinary excreta, is lessened. I have found it below the average in many instances, and sometimes, as in the case of Vallance, absent altogether. In the case published by Becquerel the uric acid amounted to .585 grammes, about the normal quantity.

The method in use for the estimation of uric acid is somewhat uncertain in its results.

The diminution of phosphoric acid is more marked than that of uric, but is not so extreme as occurs with other forms of renal disease, particularly with the waxy kidney. The diminution, though seldom extreme, is constant. It is probably owing to the loss of this acid that the urine is so often deficient in acidity. In the case reported by Mosler, already alluded to, the daily phosphates averaged 2.35 grammes, afterward 1.7 grammes. In three cases of scarlatinal dropsy already mentioned, in which recovery took place, the loss of phosphoric acid was much less decided than the loss of urea. Taking in each case the minimum, and comparing it with the amount after recovery, in the first case the amount was .499 against .89; in the second, .55 against 1.88; in the third, 1.11 against 1.5.

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[1] From Mosler, quoted by Dr. Parkes, "On Urine," p. 379.

In the fatal case of Vallance it amounted in 24 hours to only .016, an extreme and exceptional diminution.

The sulphuric acid is constantly diminished, but less so than the phosphoric acid. In the case from Mosler the daily amounts of sulphates fell to 1.7. In the case of Vallance the reduction was less than in the other constituents of the urine, the amount of sulphuric acid in 25 hours being .212.

The chlorine is variably diminished, sometimes totally absent. It is more reduced in this disease than in either of the other forms of albuminuria. The diminution appears sometimes to be exaggerated by the presence of pneumonia, to which patients suffering from this disease are liable. In the case quoted from Mosler chlorine was on one day totally absent. In the case of Vallance it was reduced to 0.17, about equalling the phosphoric acid in amount; the latter being in a state of health in much smaller quantity.

The potash, soda, lime, and magnesia are all probably diminished, but we have no sufficient data to enable us to affix the amount of diminution to each.

The chemical changes in the urine of nephritis may be thus summed up. All the constituents are diminished. The water, the urea, and the chlorides are lessened to a greater extent than occurs in any other renal disease. The phosphoric, sulphuric, and uric acids are reduced in a less marked manner, the phosphoric acid suffering most, the uric apparently least. During convalescence the constituents which have been deficient—the water, the urea, the chlorides, and sometimes the uric acid—are increased beyond the normal amount.

Albumen is invariably present, and in larger quantity than in any other renal disease. The blood crystalloids are occasionally present, and are sometimes to be detected before the albumen.

Cases.

The following cases exemplify the pathology of the disease and its general course when it proceeds to a fatal ending. Some other examples, pathological as well as clinical in their bearing, will be found in the next chapter in connection with the cause to which the disorder in each instance was traced.

Diffuse or General Nephritis, involving both Tubes and Interstitial Tissue, and giving rise to the Large, Smooth, Mottled Kidney—Severe General Dropsy, Uræmic Convulsions, Pericarditis and Serous Infiltration of Lungs—Blood Albumen and Casts in Water—Hypertrophy of Heart, increased Vascular Tension, Arterial Degeneration.

A boy seven years of age, named Richard Warren, came under my care at the Hospital for Sick Children on March 9, 1874. He had formerly had measles, but never scarlatina.

His present illness began ten weeks ago with rheumatic fever, from which he had not so far recovered as to leave his bed, when six weeks before admission he was attacked with general dropsy, which appeared first in the face and subsequently extended to the whole body, affecting the scrotum, so as to make puncture necessary. The urine was described at the same time as scanty and red.

On admission the integuments were generally and considerably œdematous; no fluid could be detected in the peritoneum, but the pleural cavities were dull below, where the breathing was feeble, as if from the presence of fluid. Bronchial râles were audible throughout the lungs. The sounds of the heart were natural; the dropsy, evidently not cardiac, notwithstanding the rheumatic history. The urine was smoky in color, and, as the microscope showed, loaded with blood. It coagulated to one-half and displayed multitudes of small granular and hyaline casts. The liver was increased in size; its lower edge could be felt midway between the ensiform cartilage and the umbilicus.

Digitalis and hydragogue aperients were given with no useful result.

On the 10th he had a series of epileptiform fits, which lasted for twelve hours; they subsided under chloroform, bromide of potassium, leeches, and baths of hot water and hot air. The convulsions chiefly affected the right side, the face being drawn in that direction.

Without following the case in daily detail the œdema continued universal and extreme, the peritoneum was distended with fluid, and the lungs were pervaded with the moist sounds of general serous infiltration.

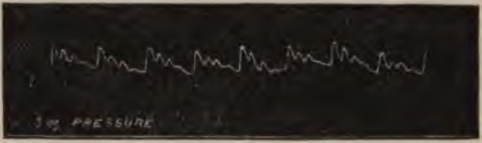
After the attack of convulsions he remained for about two days in a partially comatose condition, and then resumed his former state.

The bowels remained loose under medicine, but the dropsy persisted with little change, nor was there any marked alteration in the general symptoms. It was noticed on the 20th that the cardiac dulness was more than normal, and that the first sound was somewhat prolonged, conditions not before apparent, though the heart had been regularly subjected to observation. On the 26th the first sound was found to be reduplicated.

On April 2d Dr. Sibson kindly took some sphygmographic tracings of the pulse, one of which is reproduced on next page.

The increase of cardiac dulness was now marked; the sounds distant, the first, as before, occasionally doubled.

He had increasing distention and much pain in the abdomen and increasing difficulty of breathing. The diarrhœa continued. Without material change in his general state he quietly lapsed into coma and died on April 6th.



The urine, after the boy's admission, was regularly and somewhat elaborately examined; the daily results may be thus epitomized.

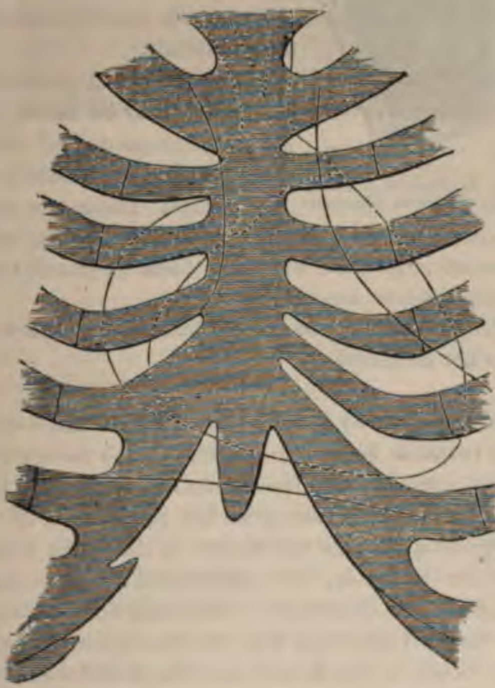
The secretion at first was smoky, with blood; latterly, though blood corpuscles were generally to be found with the microscope, it was not sanguineous to the naked eye. The color was usually somewhat deep; amorphous lithates and uric acid crystals were not infrequently present. The albumen coagulated usually to about one-half; at last nearly to solidity. The quantity of the secretion could seldom be completely estimated, owing to the loss with the action of the bowels; when it was fairly measured it varied from 24 to 26 ounces in the twenty-four hours. The specific gravity ranged from 1012 to 1020. Casts were always abundantly present, except when the urine under medicine was alkaline. The urine, when naturally acid, swarmed with casts, sometimes in such numbers as to form a conspicuous sediment. Most of them were of considerable diameter and of structureless, finely granular substance. Others of less width were either simply fibrinous or of fibrine imbedding epithelial cells. Besides the casts were numbers of unattached spherical epithelioid cells, some of which contained granules of oil.

After death the extreme general œdema was conspicuous, as in life; and as further evidence of the general dropsy the pleural cavities contained much serum, the right most.

To place the kidneys first: these were greatly enlarged, each weighing four and a quarter ounces. The capsules were thin, translucent, and non-adherent. The surfaces were perfectly smooth and "mottled," large patches being ivory-white, while others were marked with stellate and capillary injections. In section the cortex was increased in thickness to from $\frac{5}{16}$ths to $\frac{3}{8}$ths of an inch. Some parts corresponding to the white patches on the surface were nearly white, like the cut surface of a parsnip; others were injected to a reddish color, under which a white substance was visible. The cones were injected, but were otherwise little changed. The whole organ dripped with blood. There was no lardaceous reaction.

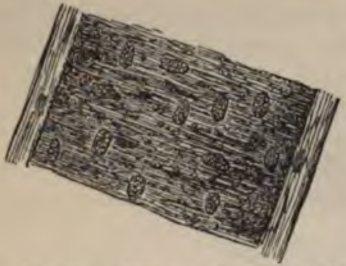
The microscope showed the tubes, both in cortex and cones, to be irregularly and greatly distended with thick plugs, chiefly composed of fibrine.

There was also a general profusion of interstitial nuclei, which were multiplied with much uniformity around the malpighian bodies and between the tubes. Around the malpighian bodies the fibroid growth generally amounted to a concentric envelope of nucleated growth of tangible thickness. Elsewhere the new formations were scarcely to be detected but by the number of intertubular nuclei. A section from one of these kidneys is represented in work on Diabetes, at page 28.



Outline of the heart in relation to the chest as exposed after death. The curved lines on either side show the extent of the lungs; the horizontal line below, the limit of the pericardium.

The heart weighed 6¾ ounces; it was of great proportionate size. Dr. Sibson kindly traced upon glass the outline it presented in relation to the chest. Both ventricles were thickened, the left most so; its walls were half an inch through, and the muscular columns within strikingly developed. The valves were healthy, nor was the lining of the great vascular cavities otherwise affected than by a couple of spots of atheroma upon the ascending aorta. A more minute examination, however, of the arterial system showed changes not evident to the naked eye. The vessels of the pia mater not quite of the smallest size showed granular and fatty degenerations of the muscular coat. How far they were absolutely thickened was a matter of some doubt, but that many of the muscular nuclei were coarsely fatty was sufficiently evident. Outlines of these vessels, showing their thickness as compared with healthy arteries, are given subsequently.


Artery of pia mater in glycerine, showing the nuclear degeneration, together with some fibroid thickening.

The additional particulars of the examination may be briefly filled in. The pericardium contained two ounces of serous fluid, and was thickly coated with recent lymph. The lungs were soaked, and the bronchial tubes filled with frothy fluid.

The liver was of large size, weighing 2 lbs. 7½ oz.; it was yellow, soft, and fatty; the surface was smooth.

The brain was exceedingly bloodless, but the cavities were empty and the convolutions not flattened.

The enormous production of tube-casts in this case—such as by themselves to form a palpable sediment—together with the extensive tubal obstruction as found after death, were enough to show that the condition of the tubes had at least a large share in the production of the symptoms. Whether the interstitial change was coeval in its origin with the tubal, or, as is perhaps more probable, was superadded to it as the disease progressed, it is not easy to determine. The interstitial change though general might have escaped discovery had not the logwood process been used.

The apparent origin of the disease in acute rheumatism is worthy of remark; probably the rheumatic products escaping by the kidneys furnished the initial irritant, rare though it be for such a cause to be followed by this result.

The symptoms—the extreme general dropsy, the bloody and albuminous urine, the cerebral disturbance, and the final pulmonary œdema—were such as belong to the tubal disorder, whether or not it be complicated with the interstitial.

The cardio-vascular changes are of interest in relation to recent discussions. That the hypertrophy of the heart dated from the renal attack was presumable from the tender years and previous health of the patient; and the circumstances of the case pointed decidedly to the view that the cardiac change was produced by the renal through the *modus operandi* of contaminated blood rather than through any secondary or associated arterial alterations. No arterial fibrosis was discernible; the vessels were as compared to the heart little altered, and that more by change of texture than increase of bulk—though some degree of muscular thickening was apparent; and yet the increase of arterial tension as recorded during life by the sphygmograph was such as necessarily to have made great demands upon the ventricle. The old view, that the change by which the circulation is retarded in such cases is not in the blood-vessels but in the blood, is enforced by this and other similar examples of renal disease in childhood, with early and considerable hypertrophy of the heart, but little arterial change, and possibly no fibrosis anywhere.

The heart in childhood becomes very rapidly enlarged. In this instance it was auscultably hypertrophied within eight weeks of the outbreak of dropsy ; while the extreme hypertrophy found after death was only a matter of ten weeks.

Tubal Nephritis in a Man of Intemperate Habits—Cramp, Purpura, Pain in Loins, General Dropsy, Urine Scanty and Albuminous, Casts, Effects of Acupuncture, Gradual Sinking.

Edward Nash, twenty years of age, a blacksmith, a well-made, powerful man, was admitted into St. George's Hospital, November 30, 1858, under the care of the late Dr. Page.

He said that for seven years he had been in the habit of drinking to excess, having commenced the practice at the age of thirteen. For the last two years he had been extremely intemperate, drinking beer, gin, and rum.

Six weeks before he came in, being at that time in apparent health, he was attacked with pain in the legs, particularly in the calves, immediately followed by an outbreak of red spots, which were described as resembling purpura. On the next day but one he began to have sharp pain in the loins ; and the urine, which had previously always been clear and plentiful, became very dark in color. Four days later his face became swollen, and the dropsy gradually spread over the whole body, occupying eight or nine days in the process. The pain in the loins now subsided, as he supposed, in consequence of his having been cupped in that situation.

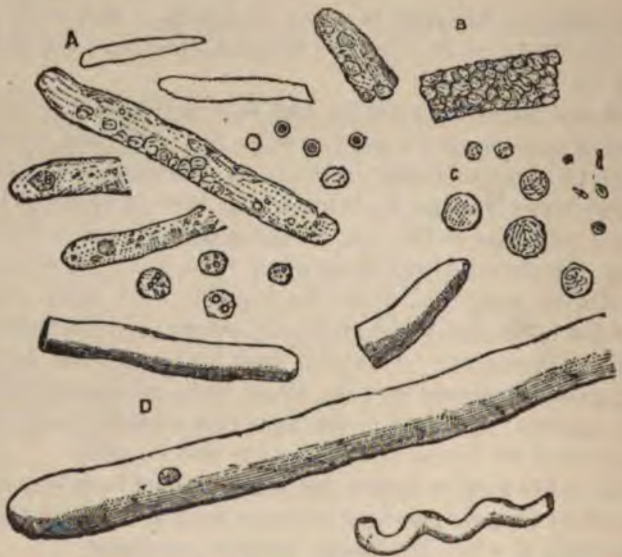
For four or five months before his attack he had been very subject to catarrhal attacks in consequence of exposure to cold, but there had been no definite exposure to which the disease could be attributed.

When he came under observation his face was white and bloated. The whole body, more particularly the legs and scrotum, was highly œdematous. He lay propped up in bed, with the right side lower than the left, and it was evident on auscultation that the right pleura contained fluid. There was evidence of fluid also in the peritoneum. There were no pains in the loins, but there was tenderness, especially over the right kidney. The appetite was good and the bowels regular, but he sometimes vomited. The tongue was clean. The pulse weak—84.

The urine was diminished in quantity and loaded with albumen, the coagulum occupying three-fourths of the tube. It was not colored with blood, though blood-corpuscles were seen under the microscope. There was also a considerable deposit of renal epithelium, some of which was fatty, while some closely resembled pus. Many transparent casts were found, some of which imbedded epithelial cells, and others specks of oil.

The frequent use of compound jalap powder, of diuretic medicines, including digitalis, and of blue pill to the extent of salivation, failed to relieve the dropsy. He was frequently cupped upon the loins with apparent benefit.

On December 9th the scrotum was punctured, and much fluid evacuated. Shortly after this it was found necessary to repeat the same expedient upon the legs. The operation was entrusted to a house surgeon, who, by way of experiment, pricked the left leg with a needle, using a lancet upon the right. Fluid escaped in large quantity. The openings which had been made with the needle healed without any bad result, while those made by the lancet gave rise to deep suppuration, pus being discharged through five of the punctures. The patient became weaker as the discharge continued, and needed stimulants, which were given. Pain in the loins was occasionally complained of. The condition of the urine, early in February, was much as before. It was scanty, acid, and as albuminous as ever; sp. gr. 1019. It contained numbers of fatty cells of renal epithelium. The casts, which in December had contained entire and broken-down epithelium, were now entirely composed of transparent fibrinous matter. They were uniform, transparent, and of large size. The fluid in the right pleura had now ob-



Urinary deposit from case of Nash. A and B, large and small hyaline, D, epithelial casts. Also scattered renal epithelium, some fatty.

viously increased, as had the œdema, and the patient gradually sank without any fresh symptom. He died on Februray 8th, retaining his consciousness to the last. The œdema had latterly been so great that the skin cracked over the legs. The abscess in the right leg had never healed, but had given rise to numerous open sores.

At the *post-mortem* examination the right pleura was found to be distended with fluid, in which flakes of lymph floated, while the lung was much compressed.

There was a good deal of sero-purulent fluid in the peritoneal cavity.

The kidneys were much enlarged, the pair weighing 23 oz. The capsules were loose and thin, the surfaces perfectly smooth and white. On section the cortex and cones were both increased in bulk, the cortex most so. The latter was nearly white, like the surface ; the cones of a pale pink color.

Under the microscope the increase of size was found to be due to distention of the tubes. The section showed that there was no increase of fibrous tissue round the malpighian bodies or elsewhere. The method of making sections in use when this kidney was examined was not efficient in the display of mere intertubular nucleation, but it could be trusted to show that there was no tangible fibrosis. The tubes were opaque and irregularly dilated. They were stuffed with fatty epithelium and loose oil-globules. In the cones some of the tubes were bare and apparently empty, while others were packed with natural epithelial cells.

The malpighian bodies were natural. Iodine gave no "amyloid" discoloration anywhere.

This case is a characteristic example of what has been described as tubal nephritis. The patient was young. The disease was probably due to the direct irritation of alcohol. It came on suddenly and ran its course rapidly—in a little under four months. The urine was scanty throughout, the amount of albumen great, and the dropsy excessive. There was pain in the loins. The case was selected to illustrate the history of the disease rather than the treatment ; but the effect of puncture upon the legs is worthy of remark. The extreme danger of making too large or numerous openings is displayed in the results which followed the use of the lancet, while the needle on the other leg did no harm. The mischief which resulted from the operation evidently contributed in no small degree to cause the death of the patient.

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CHAPTER V.

CAUSES OF NEPHRITIS CONSIDERED IN DETAIL.

COLD AS A CAUSE OF RENAL INFLAMMATION.

WITH adults, exposure to cold is the most frequent cause of inflammatory albuminuria. During childhood it is comparatively seldom that the symptoms can be traced to this cause.

In a series of 16 fatal cases in adults, which I had the opportunity of examining during life and after death, the disease was traced to cold in 8; not vaguely, but to a definite exposure of which the date could be fixed, and which in most instances had given rise to other catarrhal symptoms.

Of 54 fatal cases in children, some of which were under my own notice, others contributed by my colleagues at the Children's Hospital, the disease was traced to wet or cold in but 4.

It would be easy to collect from various sources a vast number of cases where renal disease has been attributed to this cause. Dr. Wilks, in his valuable paper on Bright's disease,[1] gives a short history, as ascertained by himself, of 22 cases; in which, either from *post-mortem* evidence or from the no less conclusive fact of recovery having taken place, the disease was ascertained to be of the kind under consideration. In 10 it was traced to a definite exposure to cold or wet. Thus, both from Dr. Wilks's histories and my own, it appears that in about half the cases of tubal nephritis which are met with in general hospitals, cold or wet, or both together, are the source of the complaint.

It will be instructive to look more particularly into the circumstances under which these often harmless agencies have become so mischievous. Here are some of the more definite instances.

A gentleman rode from Maidstone to London outside a coach in very cold weather. The same evening his wife observed that his face was flabby, and a well-marked attack of renal dropsy followed, from which he eventually recovered.[2]

A bricklayer, very much heated by carrying a great weight, drank some beer and lay down on the damp grass. Next day he was anasarcous, and three months later he died in Guy's Hospital, where he afforded Dr. Bright one of the earliest cases of the disease which bears his name (Case 4). The kidneys were enlarged, soft, pale, and apparently fatty.

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[1] Guy's Hospital Reports, 1853.

[2] Bright: Guy's Hosp. Reports, 1840.

A journeyman currier, who was of temperate habits, was often exposed to cold in his occupation while in a state of profuse perspiration. One day he was employed in washing skins, his feet being very wet. At six o'clock the same evening he became dropsical; he died in a month, and the kidneys were found to be greatly enlarged, and congested to the color of chocolate.[1]

A laborer drank a large quantity of cold water when heated and fatigued by labor in the harvest-field. He had an attack of jaundice, with "coagulable urine," and eventually recovered.[2]

A house-painter was exposed to weather, and had no food for the whole of one very cold day. In the evening he was œdematous. He died after an illness of eight months, with the large white fatty kidney.

A lamplighter was wet through for a whole week, during one or two nights of which he sat up as watchman. He "took a violent cold," and had an attack of dropsy, from which he never recovered. Five months afterward the kidneys were found to be double their natural size, white, smooth, and fatty.[3]

Dr. Wilks gives four cases, in all of which the disease had precisely the same origin. The patients were shipwrights, who were working over-hours, day and night, by the riverside. They came into Guy's Hospital with renal dropsy. The overwork, and the subsequent exposure to the cold winds from the river, probably during profuse perspiration, set up the same symptoms in all.[4]

A coachman, in the habit of drinking to excess, walked eight and a half miles in the snow; next morning he was dropsical, and in a month he was dead. The kidneys were enlarged, smooth, and greatly congested.[5]

A drunken shoemaker got wet through, and sat in his wet clothes; this was immediately followed by an attack of rheumatism, and in a fortnight by renal dropsy, which ended fatally. The kidneys were greatly enlarged, white, and smooth.

A man of dissolute habits, but in perfect health, being hot and greatly excited by drink, jumped into the Thames and swam about for some time. He immediately felt ill, and next day dropsy set in. He died fourteen weeks afterward in Guy's Hospital, and his kidneys were found to be large, white, and smooth.[6]

Several instances in which temporary albuminuria immediately followed cold bathing are reported by Dr. George Johnson in the Clinical Transactions of 1874. There were no dropsical symptoms in any of these cases, and it is probable that the renal hyperæmia scarcely reached the stage to which the term inflammation could be properly applied. But the cases are of interest as showing how constantly in certain persons congestion at least of the kidney is produced by the external application of cold.

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[1] Bright's Med. Rep., case 14.

[2] Dr. Blackall, 4th edit., p. 123.

[3] Where no reference is given, the case occurred at St. George's Hospital.

[4] Dr. Wilks's cases of Bright's disease, Guy's Hosp. Reports, 2d series, vol. viii.

[5] Case of Benjamin Patrick, p. 56.

[6] Reported by Dr. Wilks.

A child was put to bed in a newly finished house, and had an attack of renal dropsy afterward. The same result followed in the case of a woman from a similar cause.

Looking generally at the circumstances under which cold has given rise to renal dropsy, it appears that in the first place there is frequently some preceding cause of exhaustion. The patient is fatigued by bodily toil, or weakened by want of food, or he is a drunkard, perhaps under the influence of liquor at the time of the exposure.

In many cases the disease has come on under circumstances of exhaustion, where the exciting cause has been so trifling as to escape notice altogether. I may instance the case of a medical student,[1] who had a most serious attack of what must have been tubal nephritis, after a course of very close labor as demonstrator of anatomy, while at the same time he lived in a very abstemious manner.

In the same category must be placed those cases—not rare—where the disease has arisen from cold and damp affecting the body during sleep. Sleep appears to bring with it a lowering of the nervous force, which, like exhaustion or depression, allows the body to become an easy prey to influences which produce disease.

Next, there are two conditions of which both are usually present—always one. The cold is protracted, and it is applied during free perspiration. Cold, acting under these circumstances upon a person who has been exhausted or depressed, is likely enough in our climate to be followed by renal anasarca. It appears, however, that this statement does not hold good, or holds good only in a modified degree in climates either much warmer or much colder than our own.

In the accounts of Arctic expeditions, though the most intense cold was often endured, under circumstances of great fatigue, by men previously weakened by disease and hardship, renal anasarca is not among the diseases from which they suffered.[2]

Dr. Kane's men, though enduring extreme cold, exposed on one occasion for seventy-two hours at a mean temperature of 41° below zero, suffered fearfully from frost-bite and scurvy, but not from any renal affection. Other travellers within the Arctic Circle bear the same testimony as to the nature of the diseases which the climate produces, and I have been informed by those familiar with the cold districts of North America, that there renal dropsy is rare or unknown.

The companions of Franklin in his earlier expedition suffered from œdematous swellings of the limbs, but this occurred after unparalleled hardships from starvation, to which some of the party succumbed. Their chief food for some time consisted of hides, and an acrid soup made from bones. It is clear that the dropsy was not of renal origin, but depended upon the extreme state of anæmia and prostration to which they were reduced. The urine is mentioned as unnaturally copious.[1]

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[1] Related in work on Diabetes, p. 47.

[2] Kane's Arctic Exploration, ch. xvi.

Renal anasarca is not a disease of the frigid zone. The travellers in that region are exposed to far greater and more sudden transitions of temperature than are ever felt in our changeable but temperate climate. Captain Parry states that his men often underwent a sudden change of 100° or even 120°, in passing from the cabin of their vessel to the outer air, and yet none but the most trifling complaints resulted. Here we have all the circumstances from which experience would lead us to anticipate renal disease : great preceding depression, intense and protracted cold suddenly applied. From these facts we may be guided as to the way in which cold acts as a source of renal inflammation. Extreme cold, though it may stop cutaneous exhalation, probably does not allow the material to accumulate. Cold increases the action of oxygen, and gives rise to increased combustion of the solids and fluids of the body. By cold the respiratory function is exalted, and the excretion of urea is diminished. With the intense cold of the North Pole, the introduction of oxygen by the lungs is probably so great, and oxidation in the body so active, that all material susceptible of such action becomes oxidized, as much of it as can be converted into carbonic acid and water passing out with the breath. The kidneys, therefore, are not liable, as in temperate climates, to be irritated by excrementitious matter, for the stress of excretion falls upon the lungs.[2]

The immunity from inflammatory affections of the kidney which warm countries possess may be explained on similar principles : other excreting organs than the kidneys are in a state of increased activity. A failure in the action of the skin would occasion a demand upon the liver and alimentary canal rather than upon the kidneys. An exposure which in England might cause nephritis, in India would be more likely to cause dysentery or hepatitis.

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[1] Journey to the Shores of the Polar Sea. By Captain Franklin. Vol. iv.

[2] The importance of the skin as an organ of excretion, and the amount of resemblance between the secretion of the skin and that of the kidneys, may be learned from the following details :

Urea is known to be constantly present in the cutaneous secretions. It has been estimated by Funke—perhaps somewhat liberally—that 157¼ grains of this substance pass off in this way in twenty-four hours, which is about a third of what passes off by the kidneys in the same time. The same observer reckons that the amount of fluid thrown off by the skin varies, according to circumstances, from 2 to 29 oz. per hour; the corresponding variations of the solid residue being 14 grains and 107 grains. In the presence of urea, and of phosphates, chlorides, and sulphates of the alkalies, the components of sweat are those of urine.

In temperate climates it appears that the kidneys sympathize and alternate with the skin more than do any other organs. If the action of the skin is arrested, the kidneys are stimulated or irritated by the presence in the blood of the material which the skin has failed to remove. It may be that the gland will not pass the limits of healthy activity, and no harm will follow ; but under unfavorable circumstances, such, perhaps, as exhaustion or loss of nervous energy, the kidneys will be stimulated beyond their power of response, and a state of nephritis will result. We know enough of the action of the nervous system to throw light upon the fact that exhaustion predisposes to inflammatory action. Section of the branches of the great sympathetic proceeding to a gland, paralyzes the blood-vessels, and gives rise to congestion of the organ concerned. With regard to the kidney in particular, it has been shown by Bernard and other experimenters that destruction of the nerves composing the renal plexus causes the urine to become bloody and albuminous, while the kidney itself is said to be affected by a rapidly destructive process, apparently of an inflammatory kind. With the knowledge, therefore, that congestion or inflammation may result from an absence of action on the part of the vaso-motor nerves, we may easily suppose that the same changes will be peculiarly liable to happen, when from exhaustion or any other circumstance this part of the nervous system acts with insufficient energy.

The progress of the disease, when it has arisen from this cause, may be briefly considered, in so far as it differs from the general account which has been given. In grown persons a very acute and congestive form of the disease sometimes results. Examples of this form of the disorder, though less numerous than the more chronic cases, are within the experience of all who frequent a large hospital, and occur in the recorded experience of Bright, Wilks, and other writers. The following case may serve as an instance.

Exposure to Snow, followed by Œdema, with black and excessively scanty Urine —Purpura—Febrile Symptoms—Treatment by Bleeding, etc.—Gradual Accession of Coma without Convulsion—Death—Examination of Body.

Benjamin Patrick, forty-nine years of age, a coachman in a gentleman's family, had been in the habit of living an easy and luxurious life. He took meat three times a day, and drank a great deal of ale and gin. He said that he never got incapably drunk, but acknowledged that he was often "fuddled." One very cold day in January, 1854, when the snow was deep on the ground, he walked eight miles and a half in the country. At that time he was in perfect health, and had been so for years. He never had any complaint which could be referred to the kidneys. The urine had been always natural in appearance, and passed without undue frequency. The day after the exposure his ankles began to swell, and the day after that purpuric spots came out on the legs. He came into St. George's Hospital on January 11th, under the care of Dr. Page, four days after the exposure to cold. The man was of robust, plethoric aspect. The only symptoms of which he complained were the slight œdema and the spots on the legs, which were hemorrhagic in character. The pulse was hard, the tongue dirty, the bowels confined. He was put upon broth diet, he was bled to 12 oz., and was purged with calomel and senna. The blood formed a large, loose, uniform clot, nearly filling the vessel. The pulse was softer. The urine was passed with the frequent watery evacuations.

On January 13th the urine was first obtained for examination. The quantity passed in the preceding twenty-four hours was but 2 oz.; it was opaque, nearly black with blood, and of the specific gravity of 1023; it was loaded with albumen, and contained multitudes of dark coarse casts, which contained blood-globules and cells of renal epithelium; it scalded in passing. The swelling in the legs was now considerable, but soft; the hemorrhagic spots were fading. He now suffered with severe dull pain in the loins, particularly over the left kidney. He was cupped to 10 oz. on the loins, the purgative draught was repeated, and he had a pill containing blue pill, digitalis, and squills, three times a day.

Next day he was free from pain, and the dropsy was almost gone, but he complained of feeling faint, and the pulse was very feeble and slow—only 58. The urine remained the same in character, but was rather more copious.

At noon on the 16th, 3 oz. of urine were produced—all that had been made since two o'clock on the previous day; it was said that none had been passed with the action of the bowels. It was rather less brown in color, but still contained much blood—specific gravity, 1018. The casts were less numerous; they were yellowish brown in color, and contained, as before, blood-corpuscles and epithelial cells. There was also much loose renal epithelium and some imperfectly formed pus-corpuscles. The patient was very drowsy, heavy in manner, and slept much. The bowels acted profusely, with watery evacuations. The pulse remained the same. He complained of pains in the limbs resembling rheumatism.

On the 19th he seemed better, and the urine was more watery. He complained of "weight in the bottom of the belly;" the gums were spongy, and the breath fetid from the effects of the mercury. Two days later he was again drowsy, and odd in manner, with some confusion of ideas. He appeared to be passing into a state of semicoma. The urine, without increasing in quantity, was paler in color. The mercury was now discontinued.

On the 23d he said he felt nothing to complain of, but he moaned and groaned when left to himself. His movements were feeble and tremulous. The pulse was weak and quick, the skin was hot and dry, and the tongue dry and coated. A draught was now ordered, containing acetate and carbonate of ammonia, with camphor and chloric ether. He became more heavy, and on the 24th was in a condition approaching coma; he lay as if consciousness had almost departed, but when spoken to he roused himself, as if from sleep, and discoursed rationally. He breathed heavily. The pupils were contracted and remained in the centre of the orbit. The pulse was full—84. The face was puffy, but there was no œdema elsewhere. No urine was passed into the bed; what was seen was paler and smaller in quantity. Next day he was still capable of being roused, though when left alone he gave no evidence of consciousness. Breathing was very noisy, and he groaned frequently. The pulse was more feeble—74. The evacuations were passed unconsciously. Gin was now ordered, besides the ammoniacal medicine. At night he had a fit of an epileptic character, apparently accompanied with some delirium, and made so much disturbance that he had to be removed to a ward by himself. Next morning he was in a state of complete stupor, from which it was impossible to arouse him. He breathed stertorously, and uttered at intervals a peculiar cry. On the night of the 26th he expired.

At the *post-mortem* examination, which was made fourteen hours after death, the kidneys were found to be much enlarged—one of them weighed 10½ oz. The surfaces were smooth and intensely congested, but through the vascularity a buff ground-work could be seen. On section the cortex appeared greatly increased: it had a purplish color, which changed by washing to a speckled brown. Under the microscope the tubes were seen to be distended with blood and epithelial cells, with much brownish granular matter. The malpighian vessels were conspicuously injected.

The liver was hard and in a state approaching cirrhosis.

There was a small quantity of fluid in each pleura; the lungs were loaded with serous fluid, and contained some patches of extravasated blood.

The brain was wet, but not unnatural in any other respect; beneath the back of the scalp was some extravasated blood.

The following case, arising from cold and ending at least as intense congestive nephritis, presents features of much rarity; it is without parallel either in my reading or experience. The history is in some respects, particularly in the recurrence of the symptoms from cold, that of intermittent hæmaturia; but the later phase of the disease, which only I am able to describe as a witness, was that of congestive nephritis, whether consequent or not upon the other disorder, attended by an amount of congestion even to the bursting of the renal capsules.

Intense Congestive Nephritis from Cold, with Symmetrical Bursting of both Renal Capsules—The Nephritis essentially Tubal, but with the Superaddition of scattered Loci of Intertubal Inflammation, probably of the Nature of Incipient Abscesses.

A jockey, 32 years old when he died, had from his childhood suffered from attacks of pain in the lumbar region, which could always be traced to cold. With these the urine was always remarked as thick and dark, though until the last illness it was not noticed as reduced in quantity. Latterly the attacks became more severe, and were attended with sickness and shivering. These were temporary and short; they left him in good general health and able to fulfil with much acceptance his duties in connection with a large racing establishment. I could not learn that the urine had been examined medically in any attack previous to the last.

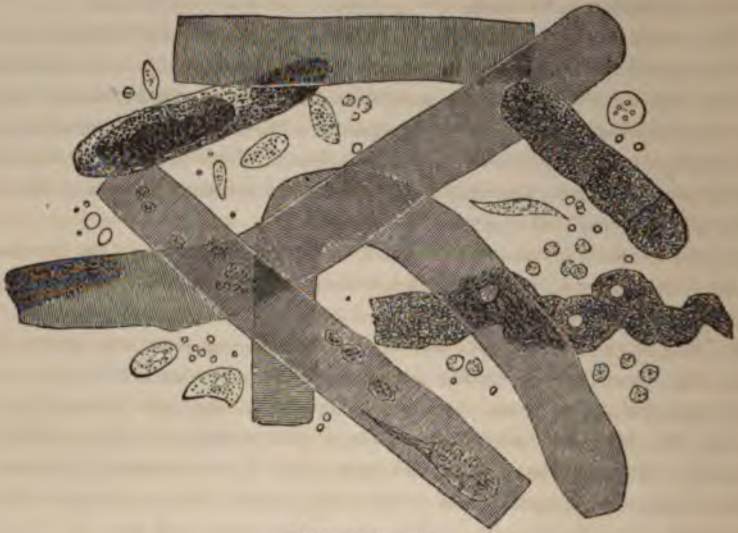
This came on six weeks before his death, when, after driving several miles in a gig at night against a cold wind, he complained on reaching home of being cold and weary, which sensations were immediately succeeded by vomiting, headache, and pain in the back. The urine at the same time became dark, and deposited a brown sediment. The illness lasted in this shape about four days, and then mitigated in all respects, save that the urine retained its brown sediment and dark color.

He then travelled, in the exercise of his resumed duties, from Yorkshire to London, and continued able to perform them in some sort for about three weeks.

On the 9th of June, 1870, five weeks from the exposure in the gig, my friend Mr. Sims was requested to visit him on account of a recurrence of the lumbar pain, and found him rolling about in bed with intense pain in this region. He had vomiting, but had no shivering, a quick pulse, hot skin, and coated tongue. It was thought that he was passing a stone, and a warm bath was ordered and a mercurial purge, which acted freely and with some relief to the pain. Mr. Sims, who next day saw the urine for the first time, found it scanty, deeply colored with blood, and abounding in blood-corpuscles and casts. The febrile state and the vomiting continued. He was now restless and occasionally wandered in mind. On the 12th only about two ounces of urine, black with blood and loaded with corpuscles, were passed; and in addition to the previous symptoms he had hiccough, tympanites, and traces of œdema about the eyelids. The vomiting continued.

On the 13th I saw him with Mr. Sims. He then had pain in both loins of an aching character. The pulse was 90, the skin now neither hot nor dry. He had frequent vomiting of bilious matter, with tympanitic distention of the belly, to which was added almost constant hiccough, which kept him awake and now constituted his chief discomfort. He had no headache; his manner was composed and his mind clear. I received three-fourths of an ounce of urine as all that had been passed during the previous twelve hours. It was chocolate-colored, albuminous to the extent of five-sixths, acid, loaded with blood-corpuscles, and swarming with thick dark casts, some blood-colored and opaque, so that their contents were not to be distinguished, but as coarsely granular; others packed with recognizable blood-corpuscles, and others consisting simply of thick cylinders of fibrine.

It was sufficiently clear that the kidney-tubes were intensely inflamed, but that this was the only condition appeared doubtful. The acuteness of the pain, its sudden access upon the journey in the gig, and the almost total absence of œdema, were enough to suggest stone in the pelvis as connected possibly with the outburst of renal inflammation. His bowels were kept loosely open; he had digitalis, together with an aqueous diet; and hydrocyanic acid, and afterward creosote to quiet the stomach, in spite of which the vomiting, hiccough, and eructation continued. The loins were cupped.



Urinary deposit.

On the 15th but 5 drachms of urine had been passed in the previous 24 hours; this coagulated to one-half, and was loaded as before with casts, though less colored with blood. The vomiting, hiccough, and eructation were still unrelieved, the œdema was still confined to the eyelids, and there scarcely evident. The patient had been restless and somewhat delirious. Dr. Owen Rees and Mr. Fuller now joined in consultation and concurred in the suspicion that the renal inflammation was associated with calculus. Tartrate and bicarbonate of potash were added to the medicine. The urine remained neither altered in character nor increased in quantity, and the symptoms persisted with little change, save increasing weakness and prostration, until the evening of the 17th, when the patient expressed a wish to walk about, while doing which he became suddenly faint and expired.

In the five days preceding his death 3½ oz. of urine were collected as the whole amount passed within that period. The characters were such as have been described; it was loaded with blood, at first to dark coffee-color, latterly only to smokiness; blood-corpuscles were proportionately numerous;

casts such as are represented were present in multitudes, and latterly there was a great quantity of apparently unaltered renal epithelium.

The absence of dropsy was remarkable throughout; none could be detected at any time but about the eyelids. There was no convulsion nor any cerebral disturbance save slight wandering at night. The prominent constitutional symptoms were those connected with the stomach, with at last rapid failure of strength.

On examination the kidneys were found to be each imbedded in a mass of coagulated blood, which lay outside their capsules in the surrounding cellular tissue. This had on each side proceeded from a rent in the capsule near the lower end of the gland, due to the kidneys having burst their investments from congestive swelling. The left kidney, which was about half as large again as the other, and the hemorrhage outside it the more extensive, was sent at once to Mr. Tuson to be drawn. The right may be described in more detail. It weighed, with the clot adherent to its outside, 10¼ oz. Its lower end was imbedded in a mass of coagulum which was not quite recent, insomuch that some portions of it were beginning to lose their color, though much was still black. The extravasation, which was adherent to the outer surface of the capsule, was about 2 inches in every direction superficially, ¾ of an inch thick in the centre, shelving toward its edges. The hemorrhage was traced to a rent in the capsule near its lower end, about 2 inches long and a quarter of an inch in width. The surface of the kidney thus exposed was roughened, and to it the clot was intimately attached.

About the lower end of the left kidney was a layer of similar clot, measuring three inches in its greatest superficial extent, and half an inch in thickness in its centre, thinning toward its edge. The rent in this instance was an inch in length and a quarter of an inch in width; it passed from the hilum along the lower margin of the gland.

In every other respect as well as in the torn capsules the condition of the two kidneys was nearly identical. The pair weighed 21¼ oz. The capsules were neither thickened nor adherent; the exposed glandular surfaces were smooth, of a deep red-brown color; they were sprinkled with dots of extravasated blood, and traversed by a few turgid vessels, neither of which, however, were conspicuous, owing to the deep color of the ground on which they were displayed. On section the cortical tissue was found to be much swollen, and to consist of a coarse intermixture of two colors, a brownish yellow and a brownish red, giving a general tone much like that of a congested fatty liver. The cones were of a deep brown color, not very different from that of the cortices; the pelvic membrane was spotted with ecchymosis.

Under the microscope it was at once evident that tubes of both kinds were enormously distended, some to a width of $\frac{1}{300}$th of an inch. Their contents were blood and epithelial cells mingled in various proportions, some containing little but blood, others wholly epithelium, and others presenting many intermediate states of mixture. The epithelial cells were generally finely granular, not recognizably fatty.

In section the state of the tubes was more fully displayed, while further and unusual changes became manifest. Many of the cortical ducts were distended to the uttermost with blood-clot, others were less strikingly but yet fully plugged with detached epithelium. These changes involved a larger proportion of the straight tubes than of the convoluted, though it could not be said that the distention was more extreme or striking in one situation than the other. The affected tubes lay among others which were empty, of which the epithelial lining was undisturbed, and which in short were absolutely natural. Besides these were other tubes which were nearly natural, no change being observable within them, save that their inner surfaces, those formed by the exposed parts of the epithelial cells, retained the traces of blood which had flowed down, but no longer obstructed their channels.

Beyond these intratubal changes others existed which were essentially extratubal. Roundish corpuscles of considerable size, such as compose unsoftened pyæmic deposits, were partially distributed throughout the intertubular region, densely abounding in certain districts to which they were abruptly limited, leaving the surrounding structure absolutely free. In some instances this corpuscular formation was seen in contact with one-half of a malpighian capsule, then abruptly ceasing, leaving the other half unaffected. In some places the corpuscles abounded for a space as evenly spread contents of the intertubular matrix, showing very distinctly as such after the removal of the tubes; in others they lay as wedge-shaped accumulations, with the base upon the capsule. The corpuscles were larger than those which belong to fibrosis; and their abrupt limitation to certain districts further hinted their connection rather with scattered suppuration than with diffuse intertubal hyperplasia. The sections here referred to are represented in work on Diabetes, page 30.

To the changes described it may be added that in some instances extravasated blood was found within the malpighian capsule, in others disposed in the intertubular spaces.

There was no vestige of a stone, or evidence of the past existence of one, in either kidney or in any of the urinary channels or cavities.

The other organs need but brief mention. The heart was flabby, the left ventricle totally uncontracted and occupied by a large globular mass of elastic, partly decolorized fibrine, from which long processes passed into the connected blood-vessels. This, probably the consequence of the failure of circulation rather than its cause, was such as forms either after death or at least after the process of dying has begun.

All the great vessels and the lungs and pleuræ were natural; and, excepting that the spleen was somewhat large and pulpy, the same statement applies to all the viscera in the abdominal and pelvic cavities, with the no-table exception which has been sufficiently detailed. The bladder contained half a drachm of pale urine. There was no sign of any external injury or internal disease which could have given rise to pyæmia, nor were there any signs of the organic results of pyæmia in any organ, unless the state of the kidneys is to be so considered.

The bursting of the capsules which took place in this instance under the inflammatory swelling was to me a new phase of disease; the pain which stimulated that of calculus was probably due to this exceptional circumstance. The kidneys may be swollen or distended in the ordinary course of renal disease to many times the dimensions they assumed in the present case, without any such result, but then the increase of bulk is gradual. It is probable that an unusual abruptness in the access of the swelling suited to its exceptionally congestive character determined the rupture.

The outbreak of the constitutional symptoms immediately upon the exposure which gave rise to them points to an analogy with intermittent hæmaturia, which is further borne out by the repeated previous recurrence of similar attacks from similar causes. But, however like intermittent hæmaturia in the outset, the course of the disease was that rather of common tubal inflammation. Blood in corpuscles was shed abundantly without—at least in the later stage, when only the patient was under observation—any of the disintegrated product which is the characteristic of the intermittent disorder. Whether or not the complaint was primarily of the intermittent type, it had merged latterly into simple renal inflammation. This was extraordinarily congestive in character, and appeared to have given rise to incipient scattered suppuration throughout the glands. This was not, indeed, evident to the naked eye, nor under the microscope were absolute pus-corpuscles detected; but the circumscribed districts of intertubal corpusculation scarcely admitted of any other explanation but that they were incipient abscesses; and in the absence of any evidence of pyæmia it could not but be inferred that these spots of collected corpuscles were among the results of the intense inflammatory action which was chiefly manifested in the tubes. The general absence of any hypernucleation or overgrowth in the interstitial tissue was clear. Abscess as a result of tubal nephritis may be said to be unknown, but with this exceptional instance in view it would seem not to be impossible.

The nearly complete absence of dropsy is worth remarking in connection with the nearly complete suppression of urine, the secretion not averaging an ounce a day, while no dropsical effusion occurred save a mere trace about the eyelids. As with suppression of urine caused by mechanical obstruction, death was due to asthenia. In connection with his having taken up to his death moderate doses of digitalis, two drachms of the infusion every four hours, the absolutely uncontracted state of the left ventricle is worth noting, as an indication that that drug had not told upon the course of the disease. With the knowledge that the *post-mortem* afforded it is probable that early bloodletting would have been of service.

The rapid and congestive form of nephritis exemplified in the preceding cases is seldom traced to any cause but cold; perhaps cantharides causes something like it. There is no line of demarcation between this and the more prolonged forms of the disease; the latter are by far the more common. The time which the disorder arising from this cause takes to reach a fatal termination varies, according to my own observation, from a few weeks to about eight months. When it ends favorably, which under judicious treatment will be the case in the majority of instances, it will mostly do so in three or four months; often in a much shorter time. When a consequence of cold, as when it proceeds from scarlatina, hæmaturia marks the commencement in the great majority of cases. With those which recover hæmaturia is nearly an invariable symptom, but it is less constantly present in those which end fatally.

The progress of the disorder is such as has been sufficiently detailed in the general sketch of the symptoms of tubal nephritis. The only point which requires especial notice is the change in the character of the epithelium which takes place in the majority of the cases. The cells have a tendency to become fatty, which is much more marked than when the disorder has sprung from scarlatina or from any other cause. In cases I have examined this was not accompanied by any obvious fatty change in other organs. The epithelium probably becomes fatty, as part of the change consequent upon the inflammatory action. That fatty degeneration should occur in an inflamed tissue is accordant with all our knowledge, though in the scarlatinal form of the disease, which has shortly to be considered, it is the rule to find the epithelium free from oil.

A more rare cause of the disorder may be mentioned as resembling external cold, insomuch as the source of the disturbance is probably the secretion due to an organ other than that which is attacked by the inflammation. When one kidney has been destroyed or incapacitated by some change confined to itself, it is not unusual for its fellow to become diseased. It sometimes happens, indeed, as will be shown hereafter, that when the primary mischief has been tubercular excavation, the remaining gland becomes, in common with other organs, affected by lardaceous infiltration as the result of the purulent discharge. But cases are believed to occur in which, after the obliteration of one kidney, the other has become the seat of tubal inflammation, with its characteristic symptoms and pathological changes. The *modus operandi* is obvious. The remaining gland is unequal to the double work, and becomes morbidly stimulated by the duty thrown upon it.

This cause of renal inflammation is, to say the least, extremely infrequent. The surviving organ usually becomes lardaceous, or simply hypertrophied, according to the nature of the change which has destroyed its companion.

SCARLATINA.

In childhood this is by far the most frequent cause of the disorder. A case of scarlatina rarely passes through its course without some trace of albumen in the urine.

It would appear that in the course of scarlatina, as well as in certain other febrile diseases, morbid products are left in the blood which the kidneys take a share in removing.

The kidneys are irritated by the poison, which selects them as a mode of exit. This view derives support from the fact, that in other febrile diseases similar results follow, and also from the observation which must have been made by every one familiar with scarlatina, that where the throat has suffered severely the kidneys are mostly exempt. The *materies morbi* may exhaust itself upon the throat, or may remain in the system as a source of further mischief. It has been thought that the kidneys become affected in consequence of the inaction of the skin, which accompanies the shedding of the cuticle ; but it appears that the kidneys are much endangered when the skin is little affected. It is, however, an important practical fact, that the action of external cold often acts as the immediate instigator of the renal disturbance. In many cases where no renal symptoms have been noticed, and convalescence has been apparently established, a too early exposure to weather has set up an attack of dropsy.

In view of the relations between scarlatina and nephritis, some experiments by Dr. Baginsky are of interest. With the object of partially suppressing the cutaneous functions he coated limited portions of skin in rabbits with impervious varnish, so as to render it incapable of transpiration, or caused it to become inflamed by the application of croton-oil and turpentine. These proceedings were generally followed by albuminuria, associated, as was afterward found, with various degrees of inflammation of the kidneys. This experimenter infers that in scarlatina the kidney is not directly affected by the poison, but suffers only in consequence of the inactivity of the skin due to desquamation. Without accepting this conclusion in its entirety the observations at least show that sympathy or rather alternation of function between the skin and the kidneys may be a potent source of evil.

It is difficult to say in what proportion of cases dropsical symptoms follow scarlatina. This appears to differ at different times and in different epidemics. The late Dr. Hillier found the urine to contain more or less albumen in about half the cases under his care during his experience at the Children's Hospital. From my own observation I should say that this statement is rather below than above the truth.

How large a proportion of children who suffer from renal dropsy have acquired the disease as a sequel of scarlatina will be seen from the fact that at the Children's Hospital, where children are treated up to the age of twelve, a series of 103 cases of albuminuria, connected apparently with renal inflammation, gave 75 where the disorder was traced to scarlatina; 3 to measles, 1 to erysipelas, 1 to acute rheumatism, and 1 in which it came on in the course of eczema; 5 from cold and 17 from uncertain causes made up the tale.

The preponderance of boys over girls holds good in scarlatinal dropsy, as in nephritis from other causes. Particulars have already been given bearing upon this point. I may add here that in Dr. Tripe's table, deduced from the Registrar-General's Report, out of 1,575 fatal cases of scarlatinal dropsy 946 were of male, 629 of female subjects. It is to be observed at the same time that the deaths from scarlatina are nearly the same for both sexes.

Scarlatinal dropsy is exceedingly rare under a year old. As an exceptional case I may mention that a child ten weeks old was under my care at the Hospital for Sick Children with dropsy and albuminuria, consequent upon scarlatina. It got quite well. This is the earliest age at which, so far as I am aware, the disease has been observed. Dr. Tripe has deduced from the Report of the Registrar-General that the deaths from scarlatinal dropsy increase gradually until the fourth year of life, when they are more numerous than at any other period. They then regularly diminish in number, until after the age of twenty they become exceedingly uncommon. Scarlatina itself causes most deaths during the third year, and it is the frequency of scarlatina, rather than the disposition to renal disease, that renders scarlatinal dropsy so common among young children. It has been shown that in a given number of cases of scarlatina dropsy is more frequent between the ages of five and fifteen than before or after these limits.

The renal affection may come on at any period after the first appearance of the febrile symptoms. It is often the first sign of illness which attracts notice, though in such a case a careful inquiry will probably show that the child has been exposed to the infection of scarlatina, and has perhaps been more or less feverish some days before. Dr. Tripe, who has based his conclusions upon a very large number of cases, states that though the dropsy may come on at any period of the exanthem, even the earliest, that it most often appears on the fourteenth day, but may be delayed even to the eighth or ninth week.[1] The experience of Dr. West leads to a similar conclusion. He assigns the second week of the disease as the most common date for the commencement of the dropsical symptoms, and believes that if delayed later they generally take a mild form.

Of 60 cases at the Children's Hospital, where the dates were ascertained as nearly as practicable, five displayed symptoms of dropsy within the first week, counting from the appearance of the rash. In 42 the dropsy began at periods pretty evenly spread between the end of the first week and of the fourth, the third week being the most distinguished in this respect. The remainder were attacked during the second month, two near the end of it.

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[1] Dr. Tripe on Scarlatinal Dropsy. Medico-Chirurgical Review for 1854 and 1855.

Speaking generally, it may be said that after the end of the first month the danger is small, but that until the lapse of the second the patient cannot be looked upon as safe.

The characters of the complaint, when arising from this cause, are those which have been described as belonging to the nephritis of childhood. The course of the disease, the secondary affections, and the mode of death, are such as have been already detailed. A point of difference appears to be in the slight tendency which the renal epithelium has in these circumstances to become fatty. It is possibly owing to this that the scarlatinal form of the disease has a stronger tendency to recovery than other forms of the disorder occurring at the same age. There is also a difference in another respect. Scarlatinal nephritis is not attended with so much congestion of the kidney as is sometimes observed otherwise. Even the most acute form of the disease does not occasion the purple engorgement which sometimes results from cold.

The smooth enlargement and characteristic white color are early produced, and are constantly found after death from scarlatinal dropsy, save where the disease has been extraordinarily protracted. Then, when the continuance of the disorder has come to be reckoned by years instead of months, it may be found that the increased bulk has shrunk and the smooth surface become granular. An instance in which the kidneys were found to be granular and contracted after scarlatinal disease of three years' standing is alluded to in the chapter on the state of the arteries (W. Smith).

And I will here relate another, for the sake of the pathological interest which attaches to it as a well-marked example of this rare sequence of scarlet fever:

Mary Whitaker died, at the age of 21, in St. George's Hospital, of chronic albuminuria, which was attributed to, and, so far as the history could be trusted, appeared to be continuous with, an attack of scarlatinal dropsy eleven years previously. The immediate cause of death was pericarditis.

The kidneys were marked examples of what has been described. The pair weighed but 3 oz. The capsules were thickened and somewhat adherent. The surfaces were beset with large irregular pale granulations, divided by purplish depressed intervals. On section the cortex was so much shrunk that little but the cones was seen. These were streaked with urates.

The microscope showed a considerable amount of intertubal fibroid growth; large tracts of cortical tubes therein imbedded and shrivelled or compressed out of tubular semblance; and certain other tubes, few and scattered, extravagantly dilated, stretched into lacunæ and crammed with epithelial cells. In addition the delicate cysts were found which are so common in connection with interstitial growth. The arterial walls were thickened.

Though it must be allowed that there was nothing in these kidneys which might not have belonged to the primarily granular or intertubal disorder, yet the rare degree of the tubal distention was fully accordant with the clinical history in suggesting tubal inflammation as probably antecedent to the protracted and fatal fibrosis.

The issue of scarlatinal dropsy in chronic albuminuria is rare ; the rule of the disease is death or recovery after a course of weeks or months rather than years; but, rare though it be, it sometimes happens that the disorder will be protracted even longer than in the cases referred to.

A boy at present 15 years of age has been frequently under my care at St. George's Hospital with uræmic symptoms connected with chronic albuminuria traceable to scarlatina seven years before. His heart is hypertrophied and probably his kidneys granular. I lost sight of a young woman at the age of 16 who had a similar condition of chronic albuminuria which had pretty clearly originated in the same cause no less than thirteen years previously. When the disorder is thus tenacious it may be generally presumed that the kidneys are granular. There is now little or no dropsy, and the symptoms in all respects are those characteristic of the granular kidney.

That scarlatinal nephritis is in most instances confined to the tubes is sufficiently shown by the frequency and usual completeness of recovery. But with fatal cases it appears that in a considerable proportion the inflammation has become complicated by intertubal nucleation or incipient fibrosis, while in some this condition only is to be found, the tubes remaining natural or affected only by pressure. Should time be given the kidney will in such cases contract and become granular, and the ultimate course of the disease will be that which belongs to this organic condition.

The following cases illustrate some of the phases of scarlatinal nephritis :

Scarlatinal Dropsy—Urine Bloody and loaded with Albumen—Pus secreted by Renal Tubes—Bronchitis—Gradual Increase of Dropsy, which became General and Excessive—Patient gradually Worn out by Suffering—Examination of Body.

Lydia Moore, a maid-of-all-work, twenty-two years of age, came into St. George's Hospital, April 23, 1856, under the care of Dr. Nairne. Five weeks previously, having up to that time had good health, she had been attacked with scarlatina in a very mild form. A week afterward she went out on a damp day, and, as she supposed, took cold. On the following morning the legs began to swell, and she afterward had severe and continuous pain in the loins. When admitted she was generally œdematous; the face was puffy and remarkably pallid; the pain in the loins constant. The tongue was coated. The urine was scanty (sp. gr. 1017) and loaded with albumen, smoky in color, from the presence of blood. Abundant epithelial casts and pus-globules were found. The œdema increased. Cough came on, with evidence of bronchitis, and there was occasional bleeding at the nose. At the end of June the œdema was still on the increase; there was fluid in the peritoneum, and dulness over the bases of both lungs, without ægophony; there was much cough and shortness of breath. The tongue was now pale, œdematous, and tremulous. The pulse small, not rapid—86. The urine had somewhat increased in quantity, and was scarcely perceptibly smoky. The casts contained pus-cells, and loose pus-cells were also found. The casts are represented in Plate III.

In the early part of July the œdema was excessive; the breathing was short, though there was now little evidence of bronchial affection. The patient was very drowsy. As time went on the œdema increased, especially upon the right side, on which the patient habitually lay. In addition to the dulness over the bases of the lungs, the præcordial region now became dull, and a faint systolic murmur became audible at the apex of the heart. The urine, examined July 19, was found to contain a considerable quantity of loose pus, and to be ammoniacal. The casts were less distinct than formerly, probably acted on by the alkaline urine. Crystals of triple phosphate were found.

By the end of July (26th) the dropsy was enormous; the cellular tissue was distended all over the body. The breathing was much impeded, and vomiting, which was now frequent, was a cause of much additional distress, for the patient could not rise from a half-recumbent posture, and with every attempt to vomit she appeared to be in danger of suffocation. The pulse was now very rapid, and was hardly perceptible. The urine was paler in color, and nearly solid when boiled. The casts, as before, contained pus globules. With the beginning of August the serous accumulation began to ooze through the skin from large visible pores, but without any sore. This exudation took place chiefly from the thighs and back, and was enough to soak four or five sheets in the course of the day. With this the œdema diminished. The urine became more and more scanty, and the patient died on the 10th, rather suddenly, as if from fainting.

The kidneys together weighed 17½ oz. The capsules were quite thin and loose, the surfaces perfectly smooth, white, and uniform, scarcely a streak of red to be seen. On looking closely the lobular markings could just be distinguished by lines a trifle darker than the rest. The lobe divisions were exaggerated.

On section the cortex was anæmic, and beautifully white, like ivory; it was increased in bulk both between the cones and outside them; the cones were increased too, but in a smaller proportion.

Under the microscope the convoluted tubes were found to be choked to opacity with epithelial cells and granular matter. The straight ducts were mostly empty, or contained only a few scattered cells; their epithelial lining having disappeared, so that only the thin membrane was left. The tubes were everywhere in contact with each other; there was no intertubal growth, nor any increase of fibrous tissue.[1]

The pleuræ, the pericardium, and the peritoneum were all distended with serous fluid. The lungs were somewhat congested. The wall of the left ventricle was thickened, the valves healthy. All the other viscera were examined and found natural.

The treatment has not been dwelt upon in this case, as the object in view is to illustrate the course of the disease. The measures used were hydragogue purgatives, and medicines given in order to relieve bronchial affection.

The nature of the complaint was clear, the fact of its having arisen from scarlatina being sufficient, of itself, to declare the existence of tubal nephritis. The attack was of a very severe sort. The intensity of the renal inflammation was evinced by the abundant secretion of pus by the renal tubes, pus globules being imbedded in the casts. The suddenness of the attack, the quick increase of the dropsy, its extension into all the serous cavities, and the comparatively rapid course of the disease, are all characteristic of the inflammatory affection of the kidney. The sufferings of the patient were, as is often the case with this disorder, very great; the only relief she experienced was in death.

The following case is a type of a most intractable form of the disease:

Insidious Form of Nephritis, following Scarlatina, with Gastric Disturbance, but scarcely a Trace of Œdema—Urine loaded with Albumen and abounding with Fibrinous Casts, but without Blood—Convulsive Attack—Death—Examination of Body.

Thomas Vallance, nine years old, was attacked with scarlatina, August 8, 1866. On the 13th he came into the Children's Hospital, under my care. The attack was slight; the throat was affected, but not severely, and the boy became convalescent, the urine being free from albumen. On the 25th, however, the urine was examined, as a matter of routine, and was found to be loaded with albumen (alb. ⅞); the urine was reduced in quantity, but was not discolored. The boy looked as well as ever; he had no symptoms which could have drawn attention to the state of the urine. There was no evidence of his having taken cold; nor had he been allowed to leave his bed.

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[1] This case has been allowed to hold its place in this edition chiefly on account of its clinical interest. Had the kidneys been examined with modern methods, interstitial nucleation would probably have been found.

The skin was dry, and was peeling somewhat incompletely. Next day the urine for twenty-four hours was obtained, and found to amount to 163 c.c., or about six ounces. There was no trace of blood to be discovered. There were few cells of renal epithelium, and a great multitude of casts, consisting for the most part of simple fibrinous cylinders, perfectly uniform and without structure. A few casts of granular texture, imbedding epithelial cells, were also seen. The child now had a heavy manner, the pupils were large, and he had been sick; but he looked well, and had no pain nor any trace of œdema; pulse 72. He was now put upon fluid diet, allowing, however, bread-and-butter; ordered to drink water plentifully, and had two drachms of infusion of digitalis every four hours. Measures were taken to move the bowels, which were obstinately confined. A little brandy was given to counteract the depressing effect of the digitalis.

On the 27th the pulse had fallen to 52, and was not quite regular; the brandy was therefore increased to two ounces daily, and the digitalis given only every six hours. The urine had now fallen to less than three ounces in the twenty-four hours, still retaining the same character. There was frequent vomiting.

On the 28th the pupils were conspicuously large, and the boy's manner unnaturally sluggish. There was a trace of œdema before each tendo-achillis. He was not pallid, and still looked and felt pretty well. The pulse was 68. He took less food, but very often vomited, especially after the water, which was therefore not pressed. The urine now amounted only to 45 c.c., or less than two ounces. Instead of the digitalis he was ordered a diuretic draught containing scoparium, acetate of potass, and nitric ether; this, however, was always vomited, as was the brandy, the water, and almost everything else. He frequently was sick, although he had taken nothing, bringing up green slimy matter.

On the 29th there was no improvement; he was dry-cupped on the loins, the slowness and irregularity of the pulse appearing to prohibit the removal of blood. The diuretic mixture was changed by the addition of nitre and squills. His manner, however, became more dull and peculiar, the pupils more dilated; no increase took place in the urine, and on the 30th, as had been anticipated, he was seized with epileptiform convulsions, a succession of which came on and caused his death in two hours and a half. They were accompanied with foaming and biting of the tongue. Excepting the trace of œdema which was noticed above the heels, there was no dropsy through the whole course of the disease. The features were always sharp, and the face free from puffiness.

The urine was not in sufficient quantity to allow of the estimation of all the components on any one day. The amount of each was ascertained, some on one day, some on another; the general results are as follows. The urea was estimated on several occasions. The date of each observation is given.

Summary of Urinary Constituents, etc., in Twenty-four Hours.

Date of observation.	Quantity in 24 hours.	Normal quantity, about a quarter the adult amount (for comparison).
August 26th to 30th...........	Sp. gr. 1023 to 1024	
" 26th to 30th...........	Quantity, 163 c.c. to 45 c.c.	
" 26th..................	Albumen = 725	—
" 26th..................	Uric acid = .0	.14
" 27th..................	Phosphoric acid = .016	0.79
" 29th..................	Sulphuric acid = .212	0.5
" 29th..................	Chlorine = .017	2.0
" 28th..................	Urea = .72	8.2
" 30th..................	" = 1.6	—

Always acid and free from blood.

Post-mortem.

The body weighed 38¼ lbs. There was a trace of œdema about the ankles.

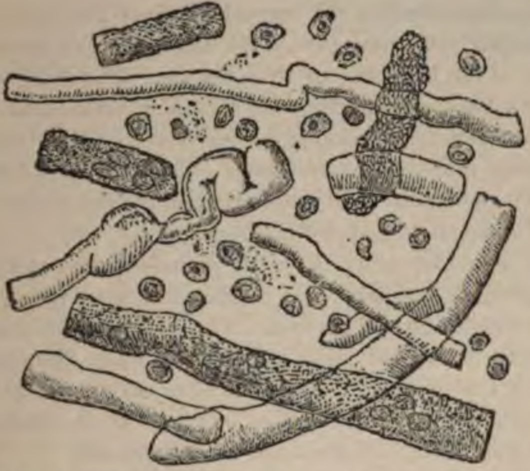
The brain and all the other organs were carefully examined; all were natural. There was no serous effusion in any of the cavities. The kidneys only require description.

The kidneys weighed 11 oz. 4 dr. The capsules were natural; the exposed surfaces were smooth and highly vascular, from the presence of a closely woven network of minute injected vessels, which gave a general pink tint. No vessel of any size could be seen. The cortex was greatly increased, dense and firm in texture. It appeared to consist of a fine intermixture of pink and buff materials, the colors being disposed much in lines radiating from the cones to the surface. Both cortex and cones dripped with blood. The cones were of a deep purple color. One of the kidneys is well represented in Plate I.

Under the microscope the epithelium was found to be natural. The part outside the nucleus was generally finely granular, a condition frequently found when no renal disease has existed.

The convoluted tubes were all filled with a fine granular material, in which many epithelial nuclei could be seen. A section hardened in chromic acid showed a general obstruction of the convoluted tubes by a fine smooth looking material, which completely plugged them up, generally lying in contact with the basement membrane, which had lost its epithelial lining. There was some dilatation of the capsules of the malpighian bodies.

The case is peculiar in many respects. From the first it was evident that it was attended with great danger. The worst cases are those in which no blood makes its way into the urine. The inflammation appeared to give rise to a copious exudation of tenacious fibrine throughout the whole tubular structure; so closing the channels that the urine, scarce able to find exit, was voided in a quantity not exceeding a tenth of its proper amount. The reduction of the urinary solids was extreme. It may be observed that the urea fell to its minimum on the day when the urine was most scanty; illustrating the general rule that the amount of the urinary elements varies with the amount of water. With the extreme scantiness of the urine the absence of dropsy is remarkable. This may possibly have been due in some measure to the removal of fluid by vomiting; though, until late in the course of the disease, it did not seem that the quantity so discharged was sufficient to make up for the decrease of urine. The alteration in the pupils and in the patient's manner, taken together with the persistent vomiting, led to an anticipation of a convulsive attack, which was verified by the result.


Urinary deposit from the case of Vallance. Loose renal epithelium, and casts containing clear fibrine and epithelium in various states.

Although this case has been inserted to illustrate the natural history of the disease, the treatment, in anticipation of what will follow, has been described in some detail, the more particularly to give an example of the failure of a plan which has been very generally successful. This case, and another which closely resembled it, are all which ended fatally of a large number of cases which during the preceding seven years had been subjected to similar treatment.[1] The rapid and extensive effusion of fibrinous matter throughout the whole gland gave a virulency to the disorder against which any remedies would probably have contended in vain.

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[1] See paper by the author. Edin. Monthly Journal, September, 1864.

The following case is given as an example of an exceedingly rare form of the disease. The scarlatinal origin was rather surmised than ascertained.

Dropsy—Persistent Diarrhœa—Peritonitis—Death—Urine free from Albumen—Kidneys well-marked Examples of Tubal Nephritis.

A child eighteen months old, named William Phillips, came into the Children's Hospital under my care. He had œdema of the face, especially of the eyelids, of the legs, and of the scrotum. The child was much emaciated and pale; the extremities were cold, the pulse rapid—132. It was stated that two months previously he was attacked with diarrhœa, which had been present more or less ever since. The dropsy had come on a fortnight ago. There was no history of scarlatina, though there appeared to be some trace of desquamation upon the legs.

The child was frequently fed with pounded meat and milk; a little brandy was given, and opiates and astringents were prescribed to check the diarrhœa, which was still present. The motions were bright green, and contained much undigested milk. The water, when obtained, was tested, with a confident expectation of finding it albuminous, but no trace of albumen could be discovered by either heat or acid. It was obtained on several occasions and examined, with the same negative result.

The diarrhœa continued, influenced very slightly by the remedies—opium, dilute sulphuric acid, tincture of the sesqui-chloride of iron, and acetate of lead—which were given for the purpose of controlling it. The child became very restless and fretful, vomited occasionally, and grew weaker. The quantity of brandy was increased to 3 oz. daily. The child became paler, and had a sunken look. He lay quiet and motionless, with an appearance of great prostration. The diarrhœa was still present. The skin was now very hot: 103.4°; and the pulse very rapid—150. The œdema had considerably extended. It was conjectured that an attack of an inflammatory nature had supervened; and the lungs were examined, and found to be pervaded by the sounds of fluid in the bronchial tubes. The belly was not tender. The child sunk a week after admission.

I was not able to see the *post-mortem* examination, but the kidneys were kept, so that I was able to examine them afterward.

The body was emaciated (14¾ lbs.) and œdematous. Nothing was noted unnatural in the state of the lungs, excepting patches of collapse. The peritoneum contained several ounces of serum, rendered turbid by pus and shreds of lymph. On the spleen there was a thick layer of lymph. All the organs that have not been mentioned were natural; there was no tubercle. It may be stated, as furnishing a standard of comparison for the kidneys, that the heart weighed 1¼ oz., the spleen the same.

The kidneys were round and full, as if swollen; they weighed 2¾ oz. The capsules had the amount of adhesion common at that time of life; the surfaces were very anæmic. On section the cortex was increased, as compared with the cones, and was anæmic and of firm, close texture. When examined in a fresh state most of the cortical tubes were seen to be closely packed with epithelial cells and granular matter; some had lost their epithelial lining, and were empty. The same conditions were made out with more distinctness in a chromic acid section, the greater number of the convoluted tubes being densely obstructed, while some were bare and perfectly empty. The capsules of the malpighian bodies were generally dilated, so that a considerable space existed between the knot of vessel and the envelope. The epithelial cells, individually, were perfectly natural.[1]

This case is of a kind uncommon, but not unknown. The absence of albumen in the urine in this form of renal disease has been reported by some writers, but I never recognized any other example. A statement has even been made by M. Philippe, of Berlin, to the effect that in sixty patients affected with scarlatinal dropsy he did not find the urine albuminous in a single case.[2] If we presume that in each of these cases the urine was tested we can only conclude that scarlatinal dropsy in Berlin is very different from scarlatinal dropsy in London. It may, however, be regarded as a fact that scarlatinal nephritis may occur and give rise to dropsy, the urine at the same time being free from albumen. The circumstance is probably one of extreme rarity. In this case all the symptoms of renal disease were present, excepting albuminuria. The peritonitis, which was the immediate cause of death, was no doubt of renal origin. No tubercles were found in the body.

OTHER DISEASES AS CAUSES OF NEPHRITIS.

There is much less to be said as to the origin of nephritis from other febrile diseases. When it follows measles it appears to pursue the same course as after scarlet fever. There are two fatal cases assigned to this cause in the series of Children's Hospital cases I have referred to, and also three in which recovery took place.

Dr. George Johnson mentions two cases of renal dropsy from this cause, one of which recovered, and the other ended fatally with inflammation of the lung and pleura. The disease appears to be identical in all respects with the form which follows scarlatina, differing only in the rarity of its occurrence.

Albuminuria, usually of a slight and temporary nature, has been found to follow or accompany diphtheria, erysipelas, typhus, small-pox, pyæmia, cholera, and pneumonia; to be produced by the vicarious elimination of bile, and as the direct result of the poisons of cantharides, turpentine, arsenic, etc. Alcohol is apt to set up the disease in a more enduring and severe form.

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[1] At the time that this and the two preceding cases occurred the logwood process was not in use; the absence, therefore, of intertubal hypernucleation cannot be asserted, though it is certain that there was no fibrosis appreciable as such.

[2] Quoted by Jaccoud: Nouveau Dictionnaire de Médecine et Chirurgie, vol. i., p. 547.

Among the disorders of the febrile type which have this tendency, diphtheria deserves mention next to scarlatina and measles. The sequence is more constant in diphtheria than with either of the forementioned diseases, though the disturbance occurs in a much milder form.

Diphtheria appears to have a tendency, even in its earliest stage, to produce albuminuria, as we must suppose from an inherent tendency in the products of the disease to irritate the kidneys. The poison, to use the simile of Dr. Sanderson, acts upon them like cantharides, which the moment it enters the system manifests its presence by albuminuria, and produces its series of anatomical changes in the kidney. Dr. Sanderson found the urine albuminous in every one of eight cases where this secretion was examined. Dr. Hillier states that of 38 severe cases albumen was present in all but five. In the fatal outbreak of diphtheria at Hertingfordbury, Dr. Sanderson states that there was only one case in which the urine was found to be free from albumen.[1]

The urine usually becomes albuminous during the height of the disease, in most cases during the first eight or ten days. The albumen may appear on the first day, rarely is it delayed until the end of the third week. In Dr. Sanderson's cases the urine was found to be albuminous within the first eight days in all but one; in this it was not so until the eighteenth. Dr. Hillier's facts give a similar result. In thirteen cases in which the urine was made the subject of daily examination there were seven in which albumen was found before the fourth day; it appeared in ten between the fourth and the ninth, in the remaining two between that date and the nineteenth. The urine usually resumes its natural character with convalescence.[2]

The secretion, when thus affected, is scanty, deep in color, and of increased specific gravity. I have found it as high as 1032. Casts are almost always present; they are transparent fibrinous cylinders, either uniform or containing epithelial cells or blood-corpuscles. In one case under my own observation the casts were dark and granular. It is stated that diphtheritic albuminuria is not accompanied by any diminution in the amount of urea and solids excreted. This, however, is very improbable.

If the diphtheria prove fatal the kidneys are found to be more or less congested, while there is some yellow opacity of the cortical tissue, due to the accumulation of epithelium and fibrinous matter in the tubes.

With renal inflammation of this origin there are usually no constitutional symptoms. I could, however, state many exceptions to this rule in which renal anasarca in an ordinary, though seldom in a severe, form has appeared. In one the albumen was more than usually persistent, lasting for three months, and accompanied with large epithelial casts. Intractable renal disease can seldom be attributed to this cause. Sometimes epithelial casts of large size are found in the urine, though there is no trace of albumen; and sometimes the casts remain long after the albumen has departed.

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[1] Dr. Sanderson: Brit. and For. Med.-Chir. Rev., vol. xxv., p. 193.

[2] Dr. Hillier: Brit. Med. Journal, 1864, p. 347.

The febrile diseases which remain to be mentioned in detail usually produce a condition of nephritis which is mild and transient, and is seldom productive of dropsy or any of the constitutional symptoms of renal disease.

Erysipelas is in a certain proportion of cases accompanied by temporary albuminuria, which comes on with convalescence, and lasts but a short time. Sometimes the disturbance of the kidneys is more serious. Johnson alludes to the case of a child who was severely affected in this way.[1]

The evidence that typhus fever is productive of a condition of nephritis rests more upon *post-mortem* observations than upon clinical examination of the urine. The disorder, usually slight and temporary, seldom attracts notice by its symptoms. Cases have been recorded, however, of acute dropsy as the result of typhus fever. Dr. Johnson gives the details of one in which the symptoms came on shortly after an attack of typhus, though it was not quite clear that the relation was that of cause and effect. Convulsions, apparently uræmic, have been noticed in connection with the same disorder.

The urine becomes sometimes albuminous with small-pox; it has been reckoned to be so in one of five confluent cases; uræmic symptoms, though usually temporary, have been known to accompany albuminuria thus engendered.[2] The same condition is frequent in pyæmic patients. After death by pyæmia the kidneys are almost always found to be in an unnatural state. The cortices are coarse in texture, firm, more or less yellow in color, and the microscope shows that the tubes are stuffed with detached epithelium and fibrinous matter.

It is well known that during an attack of cholera the urine becomes albuminous. I am not aware that the abiding symptoms of renal disease have ever been assigned to this cause. The urine becomes albuminous, and contains renal epithelium and transparent and epithelial casts.[3] In patients who die of the disease the kidneys are found to be congested, and their tubes distended with an excessive growth of epithelium. It is difficult to say whether in these cases the kidneys are irritated, as in scarlatina and diphtheria, by a morbid poison, or whether, as seems not unlikely, they suffer from the withdrawal of the watery fluid which is necessary to their function. It is probable that with insufficiency of watery fluid the elements of the urine may themselves become sources of irritation.

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[1] P. H. Bird: Erysipelas, p. 41. Dr. J. W. Begbie: Temporary Albuminuria. Edin. Monthly, October, 1852.

[2] Jaccoud: Loc. cit. M. Cartaz: Lyon Méd., September 3, 1871.

[3] Johnson on Diseases of the Kidney, p. 76.

In cases of jaundice, if the urine contain bile in any quantity, it is almost certain to contain also albumen and epithelial casts. These are generally very abundant, and exhibit epithelial cells, or sometimes almost consist of cells which are deeply colored with bile. Occasionally, even where no trace of albumen can be found, casts of this description abound. In some cases casts are present of the granular variety, consisting of broken-down epithelium. It is rare to find any constitutional symptom of kidney disease. In eight cases of jaundice under my own observation, where the urine contained albumen, there was only one where any œdema was present. In that instance the disease was cirrhosis, and ended fatally, in a boy eight years of age.

It is to be mentioned also that a form of nephritis with attendant albuminuria is often a late complication of diabetes, produced apparently by the irritation of the escaping sugar. I have discussed this question elsewhere.

It sometimes happens that during acute rheumatism the urine becomes albuminous, and it may be presumed that, as in other disorders, tubal or diffuse inflammation is established. The case of Warren, related in work on Diabetes, page 61, is an example of severe general nephritis apparently from this cause ; in the following the symptoms were transient and the inflammation probably only tubal.

Temporary Albuminuria occurring in the course of Acute Rheumatism.

Hannah Newland, twenty-eight years of age, was admitted into St. George's Hospital on February 24, 1860, with well-marked acute rheumatism complicated with pericarditis. Friction was distinct all over the heart, but the heart's sounds could be heard separately, free from murmur. There was some pain in the chest, restlessness, and much prostration ; the pulse was rapid, feeble, and irregular. She was admitted on the eighth day of the disease. The urine was found to be loaded with albumen, the clot occupying more than half its bulk ; it was very scanty, loaded with lithates, and of high specific gravity—1030. Many casts were found, some long and slender, others of large diameter. The smaller of these contained large oil-globules, the wider were granular and irregular, owing to the deposition of lithates upon them. There was no œdema, nor any pain in the loins. The patient gradually recovered. In the course of a week the albumen had fallen to one-eighth, aud no casts could be found. The patient left the hospital without any albumen in the water, and in apparent health.

In the same month of the following year the patient died in hospital of pneumonia, which was associated with a syphilitic rupial ulcer, and was apparently pyæmic. The kidneys were found to be natural in all respects, and before her death it had been ascertained that the urine was free from albumen. The pericardium was uniformly attached to the heart, which itself was of natural size and free from valvular disease.

The occurrence of temporary albuminuria in connection with acute rheumatism is sometimes due—when the disease is complicated with endocarditis—to the detachment of fibrine from the valves, and the consequent formation of fibrinous blocks in the kidneys. In such circumstances the urine becomes, as in this instance, albuminous, and contains casts. But the present case was not an example of this affection. The heart's sounds were distinctly heard, free from murmur; and the subsequent examination of the body, showing that at that time the valves were natural, lent its support to the view that they had remained intact though the pericardium had been affected. The case, therefore, was probably not one of embolism. We must suppose that either a temporary state of nephritis, such as sometimes occurs with erysipelas, diphtheria, etc., had been set up by the rheumatic poison, or else that there had been a metastasis of the rheumatism to the fibrous tissue of the kidney. Ths first view appears the more probable.

IRRITANTS FOREIGN TO THE SYSTEM AS CAUSES OF NEPHRITIS.

Among the articles of food or medicine which have the property of setting up an inflammatory state of the renal tubes alcohol deserves the first mention. It is known that this fluid, when taken constantly in excess, particularly in the form of ardent spirit, is liable to produce fibroid changes in certain viscera; but at present we have to deal with its more immediate action as an irritant of the secreting structure.

Dr. Goodfellow relates the case of a man about twenty-three years of age, who was of temperate habits, and had good health until he became barman in a large gin-shop. Soon afterward he was attacked with dropsy and albuminuria. These symptoms lasted until, at Dr. Goodfellow's suggestion, he left his calling for a time. In the course of a month the dropsy subsided, the albumen disappeared from the urine, and with it the casts and blood-corpuscles which had formerly been present; the complexion regained its usual freshness, and he seemed in perfect health. He now resumed his occupation; and notwithstanding that he observed strict abstinence in drinking, the symptoms recurred. Treatment proved ineffectual so long as he retained his occupation. He then took to a different line of life, and recovered rapidly as before, and had no further return of his complaint.[1] Dr. Goodfellow argues that the disorder had been produced by the inhalation of the vapor of alcohol, the patient having been constantly engaged in drawing and serving raw spirit.

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[1] Dr. Goodfellow on Diseases of the Kidney, p. 177.

Renal dropsy, with the symptoms of tubal or diffuse nephritis, is by no means uncommon as the result of hard drinking. It appears, however, that this effect is more often produced by a definite period of great excess—as a single protracted debauch, or a sudden change from sobriety to drunkenness—than by habitual intemperance. A young man was recently under my care with evidence of tubal inflammation—dropsy, the urine containing much albumen, blood, a quantity of renal epithelium, and epithelial casts—in whom the disease followed his obtaining a situation in the Docks, with access to the wine-casks. He appeared to have made the most of his opportunities, confessing to being drunk twice a week, and taking as a minimum a quart of wine daily. After ten months of this, without any ascertained exposure or other source of disease, he was attacked with the symptoms described. The influence of alcohol as a cause of renal disease is further considered in a subsequent chapter.

Many other substances have the property, when taken into the system, of rendering the urine temporarily albuminous, or, in other words, of setting up nephritis of a more or less mild sort. What has been described as toxic albuminuria appears to be generally of this nature. Among such substances cantharides deserves the most prominent mention, but the property of acting as renal irritants is shared by many vegetable preparations of the terebinthinate class, by phosphorus and arsenic, the irritating properties of which are manifested upon every tissue with which they are brought into relation, and by certain metals, such as lead and mercury, the action of which may be of the same kind, but as to which it is more easy to say that they make the urine albuminous than to define the process by which they do so. Most of these substances, however, which are foreign to the body, and which in part at least come within the province of the kidneys to eliminate, would seem to act with much simplicity, causing inflammatory action by their contact with the gland.

Cantharides, whether taken internally or absorbed by the skin, frequently renders the urine for a short time highly albuminous. I am not aware that œdema or any of the constitutional symptoms of tubal nephritis have ever been traced to this cause. The urine appears to acquire an irritating property, which acts most powerfully and injuriously upon the mucous surfaces, the pelvis, bladder, and urethra, while the kidneys themselves are affected in the same manner, but to a less extent. In such cases the urine contains renal epithelium and casts of the tubes, as in nephritis from other causes, though the disturbance is of a slight and temporary nature. There can be no doubt that the renal disorder thus produced is of the nature of tubal nephritis. Cases are known in which the kidneys have been destroyed by scattered suppuration as the result of this poison, but such do not come into the present question.

Turpentine appears in some cases to have an action upon the kidneys similar to that of cantharides. The urine has become albuminous and even bloody after a dose of turpentine, and the same effects have followed the taking of copaiva. As the result of either of these drugs used medicinally, albuminuria is, however, very rare, and when it occurs, transient. I am not aware that the general symptoms of nephritis have ever been associated with the alteration in the urine thus produced. Renal dropsy has, however, been attributed to the inhalation of turpentine, a process by which the organs may be acted on for a much longer time than could result from any legitimate use of the drug as medicine.[1]

Phosphorus taken in poisonous doses causes, among other results, changes in the urine characteristic of renal inflammation. Several instances have been collected by M. Ollivier[2] in the paper to which reference has been made which show that under this influence the secretion becomes highly albuminous, and sometimes contains blood and fibrinous casts. The kidneys are found, after the comparatively rapid death which ensues under the poison, to be swollen and congested, the epithelium fatty, and the cortical tubes obstructed. Many other organs and tissues share in the quick fatty metamorphosis which is caused by this deadly distilment; death ensues usually from other than renal disturbance, and I cannot ascertain that save in the urinary changes the disorder of the kidneys is attended by noticeable symptoms. The urinary changes, however, and the condition of the organ itself, are most characteristic of intense tubal nephritis.

An unequivocal condition of renal inflammation or congestion, accompanied during life by an albuminous state of the urine, has been known to ensue upon poisoning by the mineral acids, and other poisons of the irritant class must be credited with the like result.

Arsenic, whether in doses which are generally poisonous, or as medicine, is capable of producing albuminuria, dependent apparently on tubal inflammation. M. Ollivier has collected many instances of the former kind, in which œdema occurred during life in connection with the fatal action of arsenic, the urine at the same time containing blood, albumen, and casts, and the kidneys subsequently being found to be swollen and congested. The urine in these circumstances has been shown to contain arsenic, the kidneys providing one of the channels by which this poison is eliminated. Looking at the intensely irritating properties of arsenic, it is not to be wondered at that the glands in question should thus resent its passage.

It is by no means necessary that a fatal dose should have been taken in order to produce the renal symptoms; many instances have been mentioned in which they have occurred in a transitory manner as the result of comparatively small doses of the poison. Arsenic in many shapes has been known to produce the result; the ordinary arsenious acid taken into the stomach, exhalations from paint composed of arsenite of copper, and the accidental inhalation of arseniuretted hydrogen made for chemical purposes.

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[1] Goodfellow: Diseases of the Kidney, p. 40.

[2] Thèse pour le Doctorat en Médecine. Par Auguste Ollivier. Paris, 1863.

Dr. S. Weir Mitchell[1] has related instances of œdema following the use of arsenic given as medicine. In some of these cases the urine was found to have become albuminous, containing at the same time casts of the kidney-tubes. The œdema and the albuminuria came on repeatedly upon the use of the arsenic, and subsided upon its discontinuance. It therefore appears probable that a temporary state of inflammation of the kidney-tubes may ensue even from the small doses of this mineral which are used with general impunity. In some of Dr. Mitchell's cases it is to be observed that œdema occurred without the presence of albumen in the urine, a want which, as the experience of scarlatina shows, is not by any means a proof that the symptom was not of renal origin.

Nitrate of silver is another metallic poison to which albuminuria apparently connected with tubal irritation has been attributed. Under the medical use of this salt, continued for nine months, M. Lionville found the urine to be occasionally albuminous, and after death the kidneys, together with other organs, were found to be partially blackened. The cortical parts of the kidneys, especially the malpighian bodies, were sprinkled with black or blue points, and the epithelium of the tubes was the seat of fatty change.

Perhaps of all poisons lead is that which is generally the most injurious to the kidneys. Like some other metals it is largely eliminated by these glands, and it would seem that they are injured in more than one respect by the process.

The constancy with which permanent albuminuria and granular degeneration are produced by the saturnine occupations is one of the most definite facts in pathology. And many cases have been published in which a similar state of urine has been temporarily[2] produced under stress of lead-poisoning to subside with the colic and other passing symptoms. Thus, it would seem that besides a change allied to chronic inflammation in the renal fibrous tissue the metal is capable of producing a state of nephritis, which, since it is transient, must be mainly tubal, unless indeed we attribute it to some alteration of blood caused by the poison. It seems more probable, however, looking at the amount of morbid change which this metal causes in the kidneys, that the albuminuria of lead, whether of one sort or the other, is essentially renal.

Temporary albuminuria, as was known to Wells, may occur also under the action of mercury, whether due to renal inflammation or to some altered state of blood. It happens now and then that under prolonged mercurial treatment, probably for syphilis, the urine, to the surprise and dismay of the practitioner, is found to contain albumen. This causes the suspension of the mercurial course, and the secretion soon reverts to its natural state. The urine in these circumstances has been shown to contain mercury, and it is consistent with the action of other poisons to suppose the kidney to have been irritated by the unnatural material submitted to its action. At the same time I am not aware that either hæmaturia or œdema have been known to accompany this form of albuminuria, the nature of which must, therefore, remain somewhat doubtful.

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[1] New York Journal of Medicine, June, 1865.

[2] Ollivier. Loc. cit.

Thus, it appears that inflammation of the kidney may be set up by a great variety of irritants, some arising in the body, others introduced from without. The severity and duration of the inflammatory attack depends much upon the nature of the cause. The various morbid and foreign irritants which have the power of producing the disease differ much in the disturbance they produce. Thus, the matter which comes upon the kidneys as the result of a certain action of cold is more mischievous than the poison of scarlet fever, the poison of scarlet fever more mischievous than that of diphtheria. Lead and alcohol, among introduced irritants, give rise to the most protracted and dangerous form of the disease. Turpentine may perhaps come next. It is probable that one reason for the greater power for evil which lead, alcohol, and perhaps turpentine, possess is due to the continued or repeated manner in which these substances are apt to be presented to the system. Mere drugs, like cantharides, which are purely medicinal, are necessarily of transient operation, and seldom give rise to more than temporary disturbance.

Whether the attack is severe and attended with dropsy and other general symptoms, or is so slight as to be evinced by nothing else than a temporarily albuminous state of the urine, the disease is essentially of the same nature. The only difference is one of degree. Even in the mildest cases the urine contains the characteristic casts and epithelial deposit; and when there occurs an opportunity for examination of the kidney the tubes are found to be obstructed with epithelial growth.

CHAPTER VI.

TREATMENT OF NEPHRITIS.

THERE are few disorders which are more under the influence of medicine than is the catarrhal inflammation of the kidneys. Under some plans of treatment, plans which formerly were almost universally adopted, and still have their advocates, the disorder is one of heavy mortality. Under other circumstances the danger is so small that if once the complaint be recognized a recovery may be reckoned upon in a large proportion of cases. Without treatment of any kind there is reason to suppose that a large majority of the subjects of it would recover.

After the sketch which is given in the note [1] of the methods of treatment

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[1] Dr. Bright,* writing in the year 1827, and again in 1836, advises bloodletting at the commencement of the disease—"general bleeding, freely practised, and quickly repeated." But in the cases which he reports this measure appears to have been used in a discriminating and cautious manner. The same physician advises the use of purgatives, particularly bitartrate of potass, by itself or with jalap; diaphoretics, not excluding antimony, and diuretics, particularly squills and digitalis. He appeals to his own experience as testifying to the injurious effects of mercury.

Sir Robert Christison † urges bloodletting in the early stage of the disease as an indispensable measure. In the published cases bloodletting was practised to an extent which in these days appears incredible, and the results are such as to furnish the strongest argument against the precepts of this author.

He recommends counter-irritation to the loins in the form of blisters, issues, and setons, and "the general antiphlogistic regimen—which should in no case be relaxed until the force of the circulation has been broken." It appears strange that Christison, writing twelve years after Bright, should have varied from his practice in a direction which appears contrary to the progress of medical opinion. Since this date venesection has gradually fallen into disrepute, more especially in this disease, in consequence of anæmic tendencies of the complaint.

Dr. Prout, in his recommendations as to treatment, does not differ materially from the practice of Bright.

Dr. Todd, in the treatment of acute renal dropsy, restricts general bleeding to cases where its purpose is to relieve congestion of the lungs. He depends chiefly on purgatives, sudorifics (avoiding Dover's powder, the opium which it contains in his view tending to diminish the secretion of urine), and diuretics, withholding squills and cantharides in acute cases, as likely to increase the renal irritation.

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* Bright's Med. Reports, vol. i., p. 70 ; Bright's Guy's Hosp. Reports, 1836, p. 373.

† Granular Degeneration of Kidneys, 1839, p. 132.

resorted to by physicians whose opinions must carry weight, it remains to indicate the line of practice which, with our present knowledge, appears to be advisable.

First, as to bleeding. The rapid tendency of the disorder to anæmia, and the apparent association of some of the worst symptoms—the convulsions, for instance—with this condition, must be regarded as a caution against the abstraction of blood. In its power of impoverishing the blood the disease is, so to speak, its own phlebotomist; besides which the readiness with which, in most cases, the kidney relieves itself by hemorrhage, makes artificial depletion unnecessary even where it might otherwise be thought desirable. Nevertheless it must be allowed when the disorder is acute and the patient plethoric, particularly in such cases as come on from cold, where the urine is extremely scanty and the kidneys presumably congested to the utmost, that a moderate abstraction of blood, most rationally effected by venesection, may do good.

Much mischief has probably been done by purging and sweating, though in moderate measure, and in due place these means of elimination are not less than invaluable.

A general rule holds good in albuminuria, that the solid urinary constituents vary with the amount of water excreted. This suggests the importance of increasing, if possible, the aqueous part of the urine, relying on a simultaneous increase of the other constituents; and if we have regard to the mechanism of the disease the same practical maxim must follow. The urine is impeded in its exit by the obstruction in the tubes. The more completely and generally these are stopped up, the greater the reduction in the quantity of urine. It is the character of the disorder that, from causes which have been sufficiently explained, an extravagant epithelial growth takes place in the tubes, which are narrow, tortuous, and easily blocked up. To prevent dangerous obstruction it is essential that a suffi-

Dr. George Johnson,* in speaking of what he terms "acute desquamative nephritis," dwells upon the importance of external warmth. He advises the free use of diluents, proposes to act upon the skin by means of hot-air baths and antimonials, and upon the bowels by saline purgatives, jalap, or colocynth.

Dr. Bence Jones † advises external warmth—particularly when the disease has resulted from cold—warm baths, vapor baths, and warm clothing. Bloodletting, if performed at all, should be, as he thinks, from the arm, not from the loins, while the medicines advised are antimony, digitaline, and in the later stages iodide of potassium. Besides such measures to affect the general disease, hot-air baths and hard purging are advised as a means of getting rid of the dropsy; and finally iron and certain diuretics, as nitre, cream of tartar, scoparium, and cantharides.

Dr. Grainger Stewart, an excellent writer, who has given us his experience since the first edition of this work was published, mainly coincides with the opinions which are expressed in the text. He, however, has a larger trust in counter-irritants than I have expressed, and a smaller fear of the effect of cantharides as an internal remedy.

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* Johnson on Diseases of the Kidneys, 1839, p. 132.

† Med. Times, January 13, 1866.

ciency of fluid should wash out the disturbed and accumulating cells. Hydragogue purgatives and vapor baths, however necessary when uræmia is pressing, must be used cautiously, as tending to divert the water which is wanted for this purpose.

Without regarding the whole mischief as due to the blocking of the tubes—for the glandular function would probably suffer from the circulatory embarrassment were there no tubes to be obstructed—the maintenance of their channels is yet as obviously essential to the restoration of the organ as their closure would be necessarily fatal by barring the escape of the urinary excreta. If the tubes can get rid of their contents, the congestion of the gland will be relieved by secretion, the system will be cleared of its impurities; and if the interstitial tissue be not hopelessly involved the organ will gradually be restored to its healthy state, and recovery result.

There can be no doubt that it lies in the scope of the medical art to assist or to hinder this salutary process. What we can do to assist may be shortly stated.

We must insure the abundant passage of fluid through the kidney; we must avoid the use of any drugs which, under the name of stimulating diuretics, might exasperate the existing congestion; and we must enforce such diet as to reduce to a minimum that nitrogenous excess which finds its way out chiefly by the kidneys and provides in many shapes effective means of irritation. Physiological repose is to be sought, not by debarring the gland of the harmless and necessary solvent, but by cutting off the materials of urea and uric acid. "Spare diet and spring water clear" may often be found sufficient though simple remedies. Of all diuretics water is the best. It may be enough to restrict the patient to a fluid diet, with abundance of milk and a sufficiency of light broth or beef-tea; or he may take in addition some simple diluent. With children, where the kidney responds readily, the disease will often recover without further treatment.

Two or three pints of pure—best of all distilled—water may be taken daily, if the liquid food be not sufficiently diuretic, or soda-water or any other similar drink may be substituted, or the patient may take a daily portion of cream of tartar lemonade, a pleasant subacid drink, which is especially useful if there be any constipation, a condition it is always necessary to obviate.

In grown persons, or in children, when the disease is severe, digitalis is sometimes a valuable adjunct. But I have learned to doubt whether this sure diuretic is so certain not to add to the congestion of a recently inflamed kidney as I formerly supposed. It adds to the force of the heart, and I think I have sometimes traced the discharge of blood with the urine to its use. The best preparation is the infusion. The doses may vary from one to four drachms, according to the age of the patient, repeated twice or thrice in the day, or in a severe case as often as every three or four hours, until the urine has increased in quantity.

Fomentations of digitalis leaves, most conveniently applied to the abdomen, have been highly recommended, and may be tried where other plans fail, though in my own hands they have not often met with success where administration by the mouth has failed. The subcutaneous injection of digitaline—as yet, so far as I am aware, untried—is a more promising expedient.

With such measures, and even sometimes in anticipation of them, purgatives have their place. During the critical convalescençe of scarlatina it has been shown by Mr. Mahomed that the accession of renal dropsy is often foretold by an increase of arterial tension measurable by the sphygmograph, significant probably of contamination of blood and capillary resistance to its flow. This increase of tension is often sequent upon constipation, and to be removed by a purge. Purgatives, therefore, must be regarded as preventive, or at least preventively tending, where renal mischief is likely to follow upon febrile disease; and it needs no instruments of precision to enforce the very obvious lesson that if one channel of exit is apt to be injuriously affected by the passage of morbid products they should be invited to travel by others not thus susceptible. And later, in the incipience of the disease, I think a brisk aperient may help to lessen organic congestion, and perhaps relieve the system of some of the materials of renal irritation. I give a mercurial purge as often as I see a case of acute renal dropsy in an early stage. And afterward, though not relying upon such evacuants for the cure of the disorder, the bowels should be kept in daily action, with a tendency to looseness rather than the reverse. The sulphate of potass is often useful as an adjunct to remedies of a different class where an habitual laxative is needed. It is more convenient than the bitartrate, as more soluble, and is perhaps more efficient.

Formerly it used to be common to treat renal disease in a somewhat indiscriminate manner, by purging and sweating—perhaps compound jalap powder every other morning, a vapor-bath every other night. This exhausting plan was based upon the notion that the disturbed kidney, like a broken bone, stands in need of repose, a condition which was sought to be obtained by exciting a vicarious activity on the part of the bowels and the skin. But it is evident, from the want of success which attends this practice, that whatever good may be done by way of relieving the gland of its work, is more than counterbalanced by the evil which results from the misappropriation of the aqueous fluid which is needed to keep the tubes clear. It may perhaps be fairly said that the repeated use of hydragogue purgatives should be limited to obstinate and hopeless cases. If life is threatened by dropsical effusions into the pleuræ or elsewhere, temporary relief may be given by a brisk dose of elaterium, or compound jalap-powder; but this course should not be adopted until the failure of other measures has stamped the kidneys as irrecoverable.

Under such treatment as has been indicated the urine, in the great majority of cases, will increase, the dropsy diminish, and the patient pass into convalescence without the occurrence of the various secondary evils which tend to swell the mortality of the disease. After the acute stage has passed it is advisable to give iron—the perchloride, for example—at the same time lessening or withdrawing the digitalis, if that drug be in use. Subsequently acetate of potass, which may be advantageously combined with acetate of iron; or bitartrate of potass with steel wine, may be used to keep up the diuretic action. When the dropsy has passed away the diuretics may be discontinued altogether, and ferruginous medicine given, guarded with some saline laxative, such as sulphate of potash. If the urine still remain bloody perchloride or sulphate of iron appears most effectual in restoring its natural character. Gallic acid, which has been often recommended under such circumstances, is in my experience perfectly useless. Not so ergot, which, as against this form of renal hemorrhage, is an extremely effective remedy. When the urine has become copious, extra fluids, if in use, may be discontinued; and if the tongue be clean and the appetite good, as will be the case if no complications are present, more substantial diet may be allowed. If the ailment should continue, and the urine be obstinately scanty—that is, if the disorder tend to assume a chronic form—more stimulating diuretics may be resorted to: scoparium, nitre, juniper, and squills. Probably some cases occur which under any treatment will end fatally. The case of Vallance is an example of a very intractable form of the disease.

Antimony has been recommended by several competent observers in the early stages of this disorder. I cannot adduce my own experience in its favor; I am sure, indeed, that most cases will recover without it. There can be no objection to its use in a severe and recent case, particularly if other medicines have been used in vain.

Under such management as has been advised the symptoms of the disease seldom become pressing, and intercurrent disorders are comparatively rare. Should especial circumstances call for additional measures, these should not be allowed to supersede the general plan of treatment. It is of the first importance in any circumstances to insure a daily and free action of the bowels.

As to the dropsy, if so excessive as to call for mechanical relief, acupuncture may be performed. But this serious operation—for in renal dropsy it is no less—will seldom be needed. It should be done in such a manner as to secure the greatest drainage from the fewest possible punctures; made with a needle, not with a lancet.

Erysipelatous inflammation seldom fails to follow the operation when the punctures are close and numerous. It is undoubtedly true, as remarked by Dr. Goodfellow, that vesications and sores which form of themselves upon dropsical limbs are less often followed by inflammation than openings made artificially. I may draw attention to the case of Nash (p. 49), where one leg which had been pricked with a lancet inflamed and suppurated, while the other, upon which a needle had been used, did well.

The accumulation of fluid in the serous cavities will seldom require or admit of paracentesis.

For the treatment of the convulsive or other uræmic attacks it is necessary to have regard to the anæmic state of brain by which they are accompanied, and to the fact that they are apt to come on after protracted vomiting or exhaustion from some other cause. Bloodletting is generally out of the question, though now and then, if the pulse be very hard and the head hot, it may be sparingly practised. Counter-irritation is at best useless; if by means of cantharides it may add exasperation to the renal mischief. The attacks, when of the convulsive character, frequently pass off of themselves, leaving the patient much as before. The treatment must be directed toward two ends—the lessening of the cerebral irritation, and the removal of the accumulated poison which is its cause. The convulsions, if urgent and threatening, may be relieved by the inhalation of chloroform, and kept from recurring by repeated doses of bromide of potassium. Chloral may be used with a similar purpose. Probably, with these agents at our command, opium in every shape is best avoided; but nevertheless small doses of this sedative have sometimes been successful in checking uræmic convulsions. Alcoholic stimulants are generally needed, and may be given with as much liberality as the stress of the attacks demands. Such measures as have been described, however, are but palliative, and must be used with caution and judgment. But since the convulsive attacks are often attended with immediate peril to life, it may become necessary to have recourse to means which avert the present danger, though they have no permanent result. It is essential at the same time that measures should be taken for the removal of the peccant material. First in importance is the restoration, as far as may be, of the action of the kidneys. Probably, in its effect in removing urinary elements, a little urine is worth a great deal of any other evacuation. The bowels may be acted upon, even by aperients of the more active sort—jalap, croton oil, and, best of all for this purpose, elaterium; and means may be used to promote the action of the skin. Diuretics in any variety may be used, with the single exception of cantharides,[1] and diaphoretic measures may be superadded; though, with the exception of hot-water or vapor baths, they do not appear to have any marked power of lessening the uræmic condition.

Baths, when so used as to cause copious sweating, are among the most powerful correctives of the uræmic state. The hot-air or vapor bath is often successful; and recourse may be had to baths of very hot water. An uræmic patient will endure and profit by a temperature one would be afraid to subject him to in other conditions. Baths at 110° and 112° Fahrenheit have often in my experience been used only with advantage. Succeeded by an artificially heated bed they have sometimes caused sweating and relief of the uræmic symptoms when all other measures have failed.

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[1] As an example of the organic state which may be caused by cantharides given as a diuretic in renal disease, I may mention the case of a young man—not my patient—who died with symptoms of tubal or diffuse nephritis in a chronic form, and was clearly entitled to the large white kidney. Under the action of cantharides the kidneys had assumed a scarlet injection, which the underlying white only served to render the more brilliant.

Pneumonia, pleurisy, peritonitis, and the other inflammatory complications, must be treated in such a manner as not to interfere with the management of the primary disease. Mercurials, if ever of use in such inflammations, are worse than useless when these are the offspring of renal disease. The most disastrous consequences have been known to result from small doses. I might instance the case of a boy with scarlatinal dropsy whose death was caused by sloughing of the cheek produced by a single dose of gray powder—five grains. There is not the same objection to the use of antimony. Probably the more the inflammatory complications of the disease are left to external applications, such as poultices, fomentations, etc., the better. Should the patient be attacked by erysipelatous inflammation, in addition to local measures it will often be necessary to give stimulants, or increase such as are already in use.

The following cases illustrate the treatment of the disease under the various circumstances which have been described.

General Dropsy, with Albuminous and Bloody Urine, consequent probably upon Exposure to Cold—Recovery under Treatment by Water.

Eliza Crossland, fifteen years of age, an errand-girl, who was much exposed to weather in her calling, became a patient at St. George's Hospital, November 28, 1860. Eleven weeks before she had "caught cold," she said, but could not tell how. She had headache, cough, and was confined to bed. Five weeks later the water became dark, and swelling spread over the body, face, and limbs. She had pain in the loins, and the urine was passed with frequency. She had never had scarlet fever.

When admitted she had a puffy face and pasty complexion. The legs were œdematous, though less so than formerly. The pleuræ contained fluid, for there was dulness over the lower part of each, before and behind, with want of breathing and absence of fremitus. There was a short cough, with trifling expectoration; the respiration was rapid, 62 in a minute, the pulse 96.

The urine was almost the color of porter, with a considerable flocculent deposit after standing. It was acid. Specific gravity 1015. Albumen = $\frac{1}{4}$. Under the microscope numbers of casts were seen, composed of dark granular matter, probably broken-down epithelium, tinged with blood; there were others, clear and fibrinous, imbedding epithelial cells. Besides blood-globules in abundance there were cells of renal epithelium, and a few pus-cells. Dr. Bence Jones, under whose care the patient was, kindly handed her over to me for treatment. She was put upon fluid diet, which included a sufficiency of beef-tea, and she had four pints of distilled water daily, which she drank without difficulty. Under this system all œdema disappeared, and improvement took place in every respect. On January 16th the quantity of albumen in the urine was so small that it required a considerable quantity of urine and a careful use of nitric acid to detect it. There were still numbers of casts, rust-colored cylinders of granular matter, apparently consisting of broken-down epithelium mixed with blood. A few loose blood-corpuscles were seen under the microscope, though there was not enough blood to tinge the urine. The aspect of the girl was now that of health; the cheeks were quite rosy; all the œdema and pleural effusion had disappeared. Her diet was improved. A month later (February 19th) no albumen could be detected, nor could any blood-cells be found. After long search one or two casts were found of the kind seen before. She was now in perfect health, robust and strong, and able to do much of the work of the ward. She left the hospital perfectly well. She came to show herself in the following April, according to instructions, and has since remained in health. She was last seen in the autumn of 1866.

In this case the age of the patient, and the evident fact that the disease arose from cold, made the diagnosis almost a matter of certainty. She recovered completely under the use of so simple a diuretic as distilled water. The case is related as one among a great number where the same result has followed similar measures.

Tubal Nephritis from Cold—Urine highly Albuminous—Œdema—Ascites—Treatment by Water, Digitalis, and Iron—Relapse produced by a Blister—Trace of Albumen long evident after Apparent Recovery.

Edward Hill, seven years old, was brought to the Children's Hospital, September 3, 1864. A fortnight before, being then perfectly well, he was seized with cough and shortness of breath, which his parents said had come on after an exposure to cold. Four days later the face and legs began to swell, the chest symptoms subsiding at the same time.

When he came under observation the face was bloated and pallid, the legs and genital organs were swollen with œdema, the belly distended by fluid in the peritoneum. The chest was resonant, the pulse 112; the tongue nearly clean. The urine was highly albuminous (albumen $= \frac{1}{2}$).

He was put upon a fluid diet; 3 pints of water were ordered daily, and a drachm of the infusion of digitalis every six hours.

On the 11th all the œdema had gone. The swelling was only perceptible in the belly. The tongue was clean, pulse 100. The albumen was in about half the proportion before noted. The medicine was now given less often, and on the 14th, the improvement still going on, some perchloride of iron was added.

By the end of the month the albumen was reduced to a mere trace; there was still fluid in the peritoneum, though there was no trace of dropsy elsewhere. The child was pallid. The diet was now improved, meat being given every day; the water treatment was continued, but in place of the former medicine a mixture containing ten grains of acetate of potass and two of acetate of iron was given three times a day. The albumen gradually diminished, and with it the ascites, until October 22d, when no sign of the latter remained, and the child appeared to be in health in all respects, excepting that a trace of albumen could still be detected in the urine. The diuretics and the water were now exchanged for sulphate of iron, and afterward gallic acid, but the trace of albumen remained, the child all the time being, as it seemed, perfectly well. In December (14th) it was determined, though not without misgiving, to try the effect of counter-irritation. A blister was put upon the loins, and the sore kept open by means of blister ointment. The immediate effect of this treatment was a great increase in the quantity of albumen in the urine. After a time, under such measures as had been at first adopted, the albumen again became reduced to a barely perceptible quantity; this remained without change, the health of the child being perfect. On January 18th he was dismissed in this condition. He came back to show himself on the last day of the following February, by which time albumen had ceased to be evident to chemical tests. He was in perfect health.

The attack, one of inflammation of the tubes of the kidneys, came on apparently from cold, and in consequence presented a less promising aspect than if it had arisen from scarlatina. As in many cases of the same sort, a trace of albumen lingered long after the patient was well in every other respect. Gallic acid, as is usual in such cases, proved useless. The injurious action of cantharides in the inflammatory form of albuminuria, whether applied internally or externally, was displayed by a great increase of albumen after the application of the blister. The final removal of the last trace of the disease was due to time, not to treatment.

Scarlatinal Dropsy—Treatment by Water, Digitalis, and Iron—Recovery without Complications.

William Floyd, one year and nine months old, came under my care as an out-patient at the Children's Hospital, on December 19, 1861. Three weeks before the rash of scarlatina had appeared; a fortnight later the belly, and then the face and legs, began to swell. The bowels were loose.

When seen he was puffy and pale, with elastic swelling of the limbs, which did not "pit," a very usual state of things with children. The belly was swollen and fluctuated; the urine smoky and intensely albuminous.

He was ordered half a drachm of the infusion of digitalis, with two minims of tincture of sesquichloride of iron, three times a day, and to drink in the course of the day two pints of spring-water besides his ordinary fluids.

When seen on the 23d he was no better; the urine had not increased, and on inquiry it was found that he had not drunk all the water. The digitalis and the tincture of iron were doubled in quantity; he was ordered to be strictly confined to liquid food, and the water was enforced. At the next visit, on the 26th, all œdema had gone, though the belly still contained fluid. The urine was still bloody, but was much less albuminous. The same system of treatment was persisted in. On January 19th all dropsy had disappeared; the urine contained no blood and only a trace of albumen. Under the action of sesquichloride of iron this rapidly disappeared; the child returned gradually to his ordinary way of living, and was dismissed in perfect health. Neither œdema nor any trace of albumen could be discovered after January 9th.

The case illustrates the treatment of the uncomplicated disorder.

Scarlatinal Dropsy, with Convulsive Attacks—Recovery.

George Taylor, three years of age, had an attack of scarlet fever, which was followed by swelling of the face, legs, scrotum, and belly. Diarrhœa came on and the swelling subsided. The bowels remained loose after the swelling had entirely disappeared. He was then attacked by vomiting, which was frequent through the whole of one night, and in the morning three well-marked epileptiform fits occurred in succession. Between the fits, and subsequently, he was drowsy. He was now brought to the Children's Hospital and became my patient. He was extremely pallid, but without dropsy, except that the face was puffy. The head was hot, the tongue coated, the pulse rapid, 163. The urine was scanty, the color of dark sherry; when boiled the clot of albumen occupied half the bulk of the fluid. He was ordered to be fed entirely on fluids, including strong beef-tea, to drink two pints of spring-water daily, and to take half a drachm of the infusion of digitalis, with half a minim of laudanum, three times a day. He had no more fits. The urine increased in quantity and gave a copious deposit of epithelium and epithelial casts. When he had been under treatment for three days all chance of head symptoms appeared to have passed away, and the tincture of sesquichloride of iron was given instead of the opium. On the fourth day the albumen was reduced to a hardly perceptible trace, and on no subsequent examination could any be discovered. Within a fortnight of his coming to the hospital he was in perfect health.[1]

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[1] Several illustrations of the same method of treatment are given in a paper in the Edinburgh Monthly Journal for September, 1864.

CHAPTER VII.

GRANULAR DEGENERATION—MORBID ANATOMY.

The external features of kidneys affected by this disease are so obvious that they cannot be mistaken, while the minute changes by which the outward form has been modified are no less evident to microscopic examination.

In using the terms "granular" and "granulation," it must be understood that they refer to the state of surface, as to smoothness or the want of it. A granular kidney is one of which the surface, when the capsule has been removed, instead of being level and smooth, has upon it little projections, each of which forms the segment of a sphere, and which have been described as granulations.

Some kidneys there are which, when stripped of their capsule, remain smooth and polished, but which appear to contain in their substance specks of white matter.[1] These have sometimes been described as granular kidneys, but are not granular in the sense in which the term is here used, and are totally distinct pathologically.

In kidneys which have acquired granular outsides, there has invariably taken place a certain definite change in the fibrous tissue by which the tubes are separated. Of this the alteration of surface is at once the result and the proof. The change is closely analogous to that which produces cirrhosis of the liver. A morbid increase in the intertubular tissue of the organ begins at certain points upon the surface and extends inward. The new growth, as it is formed, contracts, and in contracting not only encloses and compresses such parts of the gland as are in its path, but draws in the surface at its point of origin. This, taking place at regular intervals, results in alternate elevations and depressions, or, in other words, in granulation, the size of the granules being regulated by the distance between the starting-points of the new formation.

The obvious changes to which this process gives rise may be shortly sketched, before proceeding to the more minute details which will rest on microscopic evidence.

Early Stage of Granular Degeneration.

The first change that we can recognize as a result of the disease is unevenness of surface. The capsule is, perhaps, more adherent and thicker

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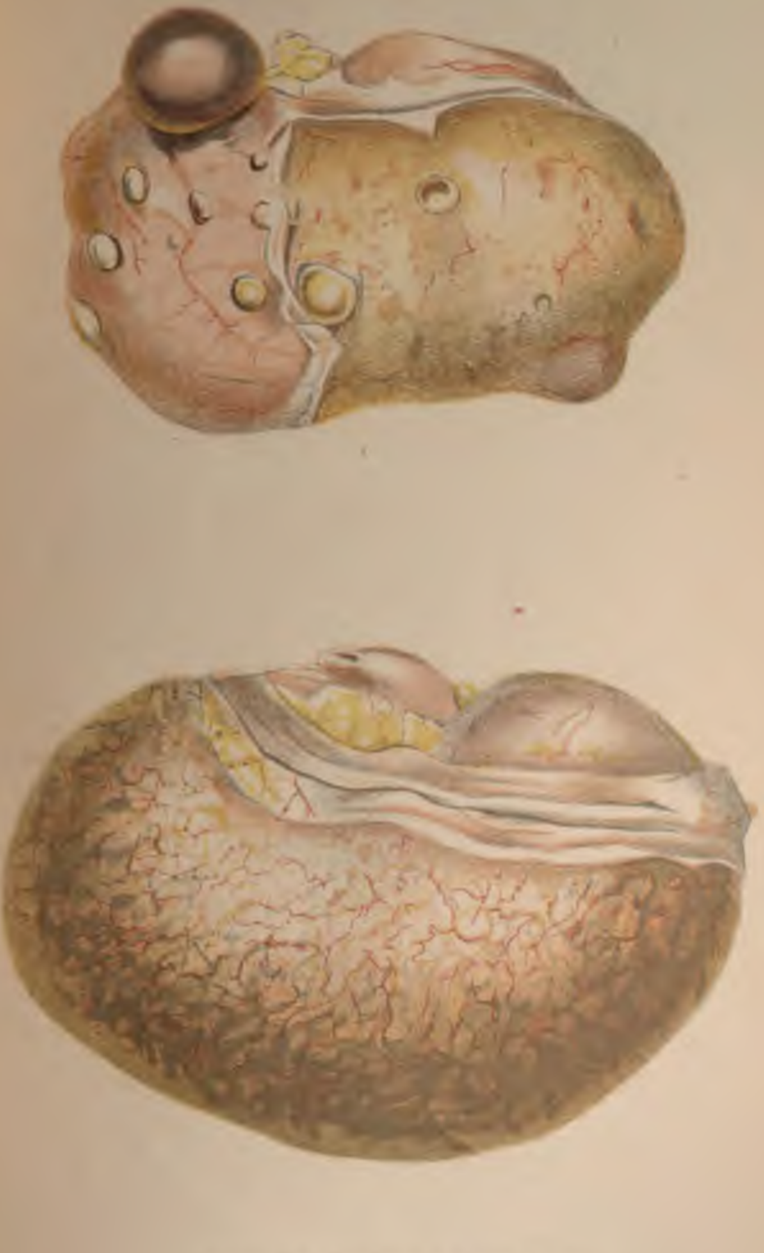
[1] See work on Diabetes, p. 27.

PLATE IV., *to face page* 94.

A pair of Kidneys affected by Granular Degeneration. They are of unequal size. The larger has had the capsule removed, so as to display the regular arrangement of granulations beneath. The smaller, which has the capsule partly removed, shows a number of cysts projecting from the surface. As not unfrequently happens, the contracting process has gone on unequally in the two kidneys, giving rise to the greater number of cysts where the shrinking has been the most. The outsides only have been shown, since it is by the state of the surface that granular degeneration is chiefly characterized. Had the organs been shown in section, the most noticeable fact would have been diminution of the cortical substance.

The kidneys represented were taken from the body of a well-known jockey, who died at the age of forty-eight, having had albuminuria, dropsy, and obstinate vomiting, followed by coma. He had had gout. At the post-mortem examination the kidneys were in the condition depicted, with extreme shrinking of the cortices, evident on section. The left ventricle of the heart was hypertrophied. The brain was anæmic and wet, and there was an incrustation of urate of soda upon the cartilages of the great toes. With these exceptions all the organs were natural.

Plate 4



PAIR OF GRANULAR KIDNEYS

than natural. On removing it some part of the surface has lost its even curve and is beset with little half-formed projections of small size and almost inappreciable prominence. Often between some of these granulations veins may be detected, visible to the naked eye, and having somewhat of a stellate arrangement. The color of the organ is not at first much altered; it is perhaps of a somewhat reddish or congested tone. On section such a kidney could hardly be distinguished from one in health; it may be that the cortex has a slightly red color and coarse grain, and possibly one or more small cysts may be detected in it. It is not much altered in bulk. In texture it is perhaps harder than natural. Such are the more obvious characters of a kidney in an early and incomplete state of granular degeneration.

If a section of such a kidney be examined with the microscope we find little fibrous processes, starting inward from the depressions, which often imbed shrivelled tubes. Perhaps such contracted remains are all that indicate the path of the new growth. The deep parts are as yet free from any change, and the majority of tubes natural in all particulars, though here and there one may be seen packed with granular matter, or clear fibrine. The epithelial cells are natural in all respects. It has been stated that this disease depends upon a crumbling or disintegration of the epithelium; but I have never been able to find any alteration in the cells, excepting now and then a deposit of oil in their outer part—a change which, as has been intimated, occurs in all varieties of renal disease, and also in health, together with such distortions as clearly result from pressure.

It may be necessary to add to these statements that such kidneys as have been described as in this early condition of the disease have been obtained from persons who have died of some disorder not renal. There is as yet neither dropsy nor albuminuria, nor any change by which the incipient disease can be recognized.

Thus granular degeneration generally begins in a slow and insidious manner, the departure from health being most gradual and indefinite. But another mode of origin, though comparatively an infrequent one, is to be recognized. It is occasionally a sequence of the diffuse nephritis which has ensued possibly upon the continuance of tubal disturbance, or may have begun in association with it, attended with symptoms of acute renal dropsy. This result of the inflammatory disorder which is sometimes exemplified in connection with scarlet fever has been dwelt upon in a preceding chapter.

The large white kidney, which to the naked eye may give no evidence of other than tubal change, will in certain cases, if effectively displayed in section, show a general intertubal growth of connective-tissue nuclei. This, if the patient live long enough—which he seldom does—will gradually lead to fibrillation and contraction, and eventuate after the lapse of years in shrivelling and granulation, not materially different in appearance or nature from those which creep upon the healthy kidney by the slow steps of primary and chronic fibrosis. The large white kidney about to undertake this process, shows at first a few dimples on its otherwise smooth surface, which deepen and multiply until at last, in comparatively rare instances, all traces of the original white swelling have been replaced by atrophy and granulation. Perhaps more often, if such be the origin of the disease, some proportion of the cortex—the deeper more likely than the superficial—will retain the characteristic whiteness and still give evidence of increased bulk. But time may in the end entirely efface the early tubal history and write upon the organ no record but of intertubal and fibrotic change.

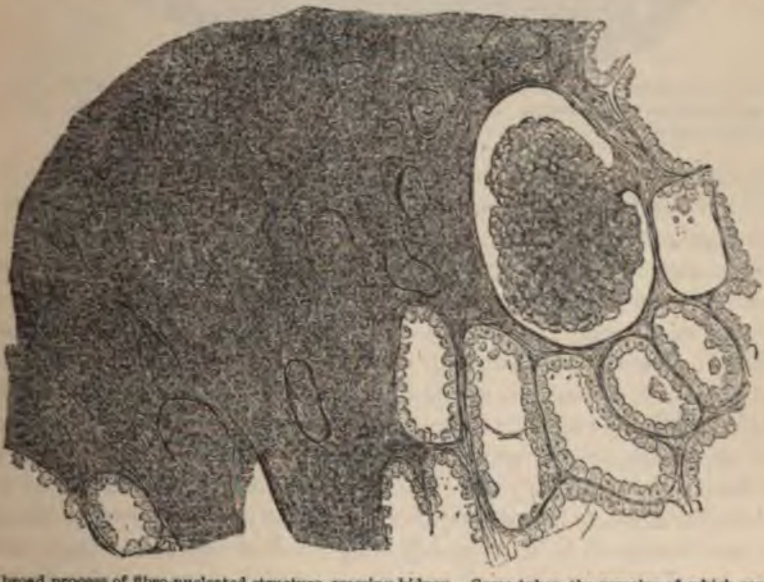
It is certainly the rule for the large white kidney of renal inflammation and acute dropsy to display, if examined adequately, the more evidently the longer it has lasted, more or less interstitial nucleation, or even general thickening of the intertubal fibrous tissue, slight, but sufficiently appreciable. The formation of new nucleated tissue may even be so early and abundant that the affection might as fairly be regarded as acute fibrosis as diffuse nephritis. Such a kidney may be greatly enlarged, mottled, and as yet smooth. The contractile tendency which it has is latent, perhaps never to be declared. The most conspicuous change may be profuse nucleation with some increase of bulk of the connective tissue. Some tubes are plugged, as in purely tubal nephritis, some are altered by pressure from without, while probably others are perfectly natural. This form of the disease begins suddenly from a definite cause, and is early fatal by way of acute renal dropsy. It has its large and clinical analogies with diffuse nephritis: according to minute morbid anatomy it would be classed with the granular kidney.

ADVANCED STAGE.

The appearance of kidneys which have reached an advanced and well-marked condition of granular degeneration is very characteristic. They are usually reduced in size, sometimes so much so that a kidney which should weigh five or six ounces may weigh only two or three. The longer the disease lasts the smaller the kidneys become. In some cases the organ has its natural weight, or is even rather increased. Sometimes the disease appears to have progressed faster in one kidney than the other, causing an inequality of size.

The shape has lost its regularity in consequence of the shrinking having taken place unevenly; often the central portion seems to have fallen in more than the ends. The capsule is generally thickened, opaque, and adherent, though it will generally come off without tearing up the substance. The surface exposed is studded with prominent hemispherical granulations, often about $\frac{1}{16}$ of an inch in diameter, though they may be either larger and smaller than this. These granules usually have a light color like that of parched peas, while the depressed spaces between are tinted with vascularity, so that they have a purplish or faint red color. The superficial vessels are only seen in these intermediate spaces where an irregular network often exists, forming a contrast with the prominences, which are always bloodless. Cysts are often conspicuous on the surface. When a section is made through the centre of the gland the cortical part, especially near the surface, appears to have undergone most alteration. Often the part lying between the cones and the capsule is reduced to the thinness of a shilling. In color and texture it has undergone much alteration. The natural brown color has been replaced by a yellowish gray or buff, which often appears closely mingled with a red tint, as if two materials, a red and a yellow, were closely intermixed. The grain is closer and firmer than in the natural state. Cysts are often found both in the cones and the cortex. The cones are less affected than any other part of the organ; when altered they assume a yellowish or buff color, approaching that of the cortex. A pair of kidneys affected as described are represented in Plate IV.

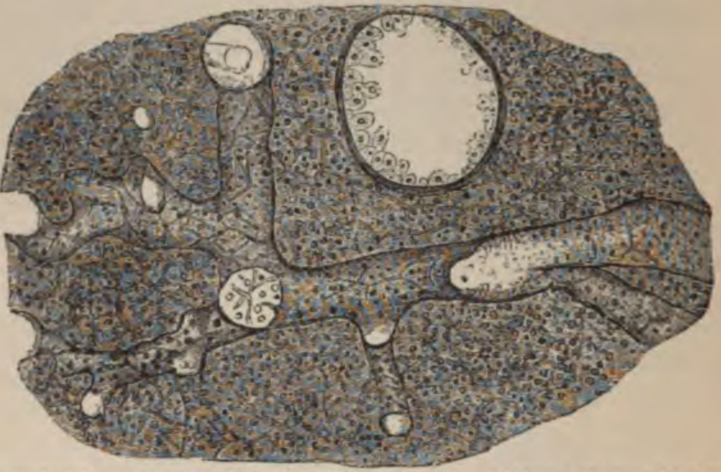
When the disease has arisen from long-continued renal congestion, as the result of heart disease, the kidney is larger and presents a much redder color, both within and without, than when it has come on from other causes. The granulations are smaller and more indefinite, and the distinction of color is less marked between the granulations and the depressions. The organ is harder and more brittle, and is often loaded with blood, which obscures its structure, so that it is not until the injection has been removed by soaking in water that the buff color of the cortex is apparent.



A broad process of fibro-nucleated structure crossing kidney. Some tubes, the remains of which can be traced, are involved in it, while others have entirely escaped.

The anatomy of the diseased organ can only be made out in translucent sections, which display all the parts in their natural proportion and relation to each other. It is necessary that the preparation should include the capsular edge.

The logwood plan, perhaps, answers best. A section made from a portion of the fresh tissue congealed in a freezing mixture will answer the purpose, or from a part hardened by boiling. It was by the latter method that the intertubular formation was first demonstrated. The fact that the same appearances are even better shown by other methods of preparation is an answer to a suggestion which has been made that the alterations in question were produced by the process employed.[1]



Fibro-nucleated growth traversed by new capillaries, from a granular kidney in the case of a child. The circular cavity is a tube in section. This woodcut and that on preceding page refer to the case of Tillet related subsequently.

The microscopic appearances in advanced granular degeneration are as follows: There is an obvious growth of fibroid tissue in the organ—not old fibre such as could be attributed to any falling together of the skeleton of the gland upon atrophy of its secreting elements, but a highly nucleated and apparently actively growing structure, as different from the framework of the healthy organ as youth from age. And it is to be added that where this material exists in bulk, capillaries, evidently of new growth, are sometimes to be traced in it. The adjoining woodcut represents such as seen in a broad fibroid process, in an instance of precocious granular degeneration elsewhere related more fully. Delicate vessels in abundant and regular arrangement traversed large masses of the fibro-nucleated growth far away from any of the original structure of the organ.

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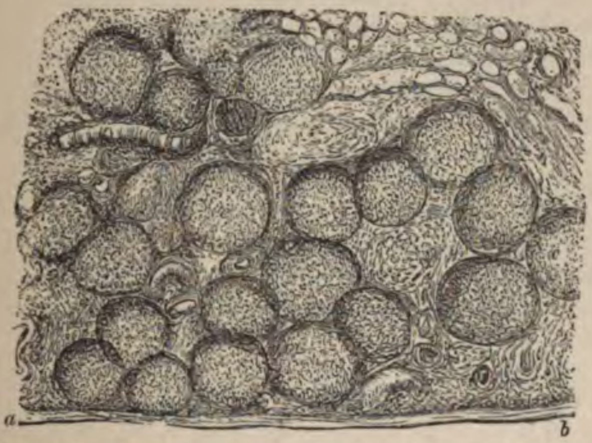
[1] Med.-Chir. Trans., 1860. Paper on Diseases of the Kidney Considered in Relation to their Origin in the Tubes, and in Intertubular Structure.

The superabundant fibrous tissue is usually to be seen around the malpighian bodies and the blood-vessels, so that when either are crossed by the section a thick fibro-nucleated ring is seen surrounding them. The most conspicuous alterations are immediately under the capsule, which itself is thickened and adherent. At the points of superficial depression streamlets of fibroid tissue pass into the organ imbedding the malpighian bodies, which retain their size, and the tubes which become compressed, to mere microscopic threads. As the nuclear material extends inward it becomes diffused, and spreads over and between all the tubes in its vicinity. The growth may be seen opposite each depression, as in Plate V., of visible breadth; or its existence may be chiefly indicated by the contraction it has caused, so that an angular space under the capsule is seen filled by the shrivelled remains of tubes. The malpighian bodies in such cases are aggregated, owing to the contraction of the parts between them, while they themselves resist the compressing agency. (See woodcut.)



Section of an advanced granular kidney from a gouty subject, showing the destruction of tubes consequent upon the contraction of the fibrous tissue, and the consequent aggregation of the malpighian bodies. *a b*, capsular surface; *c c*, malpighian bodies. A few tubes are seen at the deeper part of the section.

The new fibrous growth which is the essence of the disease is sometimes seen in isolated patches of some extent, in the deeper parts of the cortex, but this is comparatively uncommon. Some portions of the cortical tubes have been reduced to mere threads, through which no passage remains, while in other places they are irregularly dilated. They are not uniformly affected. It is a character of the disease that the increase of fibrous tissue takes place, not unevenly throughout, but at points a little removed from each other and apparently determined by the position of the blood-vessels. The tubes in the track of the growth are involved while others escape. Thus it is common, particularly in the earlier stages of the disorder, to find many tubes perfectly natural. Those that are altered are in one of two conditions, both of which are generally found in the same kidney.

Some are irregularly packed with their epithelial growth. This condition is not uniformly spread through the organ. The accumulation of epithelium is much less decided, as well as less general, than with tubal nephritis. In many cases some of the ducts are distended with dark granular matter, probably the result of the breaking up of the epithelial cells, subsequent to their detachment from the wall of the tube. This detachment and accumulation of the cells may perhaps be explained by supposing them to have become undermined by the fibrinous exudation which is poured out by the tubular membrane.

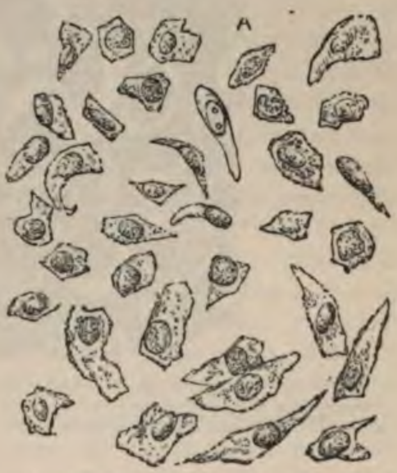
Besides the tubes thus affected are others in a different condition, which is a constant accompaniment of the disease. Many of the tubes are occupied by a transparent fibrinous material, which has taken the place of the epithelial lining. This material is sometimes quite uniform, transparent, and glassy; it is sometimes studded with oil-globules, the result of a degenerative change in itself. Sometimes this matter, probably by being forced along the windings of the tube, becomes broken up into very fine fragments, and may eventually pass out with the urine as dark, coarse, granular casts. Such casts display at first no structure; they look opaque and granular, but if touched with acetic acid they lose their obscurity and show the broken pieces of fibrine of which they chiefly consist.

What has been said with regard to the convoluted tubes will apply almost word for word to the tubes which form the cones. The contents of the latter channels exactly correspond with the casts found in the urine. They are usually filled with transparent fibrinous matter, sometimes with dark granular plugs, while sometimes they contain more or fewer entire cells of cortical epithelium.

While dwelling upon the morbid anatomy of granular degeneration of the kidney it may be well to add a few words upon the condition of the epithelium. It is the more necessary to do so, as Dr. George Johnson formerly thought, and I believe still thinks, that the change is primarily due to a crumbling or disintegration of the epithelial cells. I have examined these cells in a great number of granular kidneys, and have carefully drawn their outlines and dimensions as seen through a $\frac{1}{8}$-inch object-glass. The conclusion I long ago formed, one which has been justified by careful and continued observation, is this: In the vast majority of cases, in all cases excepting those in which the contraction of the organ has become extreme, the epithelium is exactly such as is found in healthy kidneys. When changed, it is by an alteration in its regularity of form, becoming somewhat angular, as if cramped in growing space. In one or two cases it was found that some cells had become shrivelled, much as the cells are found to be reduced in advanced cirrhosis of the liver. These changes, slight as they are, are never found excepting in very advanced cases. The cells may, of course, from various causes, contain oil, but they do not do so more often than do the cells of kidneys which appear to be natural, or which are affected by other morbid changes.

In the more advanced stages of the disease the granular kidney almost always contains cysts, sometimes of considerable size, sometimes microscopic. Cysts are found also in connection with the lardaceous disease, as will be hereafter seen, but they never occur in consequence of pure tubal nephritis.

Cysts are found both in the cortex and in the cones. Although frequently conspicuous objects to the naked eye the majority of them are very minute—mostly of a diameter but little more than that of the tubes among which they lie. They frequently have a somewhat linear arrangement, particularly evident in the cones, where they are frequently oval in shape, and lie end to end like a string of sausages. The walls are composed of thin membrane, upon which nuclei, or even epithelial cells, are sometimes seen, and which closely resemble the walls of the neighboring tubes. From these facts there can be little doubt that the cysts are produced by the transformation of tubes. In order that this should take place it appears to be necessary that there should be an intertubal development of contractile tissue. By this means the tubes are narrowed where they have become involved, and are even completely obstructed by the external pressure, as by ligature, at certain points. The occurrence of these cysts may be looked upon as a proof of the existence of such an interstitial formation. Within the parts so cut off fluid accumulates until cysts are produced, which are most numerous where the atrophy of the tubes is the greatest.

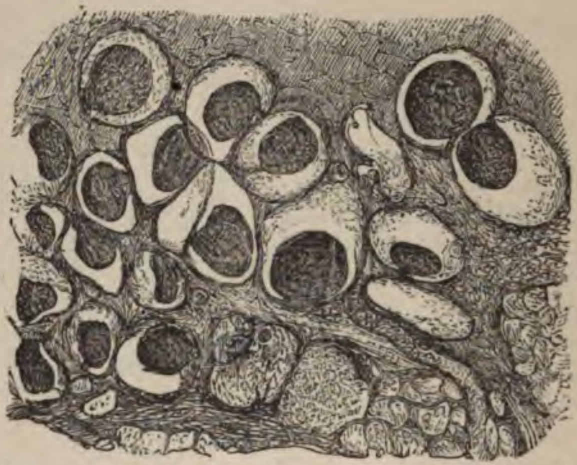


A. Epithelial cells from advanced granular kidneys, distorted by pressure.

Besides the cysts thus formed there is frequently seen in this disease a peculiar dilatation of the capsules of the malpighian bodies, which become converted into cavities of a globular shape. An empty space remains between the wall and the capillary knot, which hangs like a pea in its pod, by a stalk from one side. It is generally reduced in size, and distorted in shape, as if from pressure. The cysts thus formed are often visible to the naked eye, giving a fine spongy appearance to the part of the cortex affected. They probably owe their origin to the occlusion of the tubes in the manner which has been described. The fluid poured out of the malpighian vessel is obstructed in its way out, and accumulates in and distends the capsule.

Thus, cysts are produced either by the subdivision of the ducts or by dilatation of the malpighian body. Both proceed from the same cause, namely, from the obstruction of the tubes by external compression.[1]

Before leaving the subject of cysts as depending upon intertubal formation it may be pertinent to state that, as far as I have been able to observe, the enormous cystic kidneys which sometimes occur result mainly from an extravagant extension of the process which has been described. When the kidneys have been apparently transformed into a collection of large cysts, however great the increase of size may be, the microscope generally shows that the renal structure which remains is altered in the manner characteristic of granular degeneration; and it may be added that the symptoms in such cases are such as belong to that disease.



Section through the cortical part of an advanced granular kidney from a gouty subject, showing the transformation of the malpighian bodies into cysts. The capsules are dilated, the vascular knot compressed, while fluid has collected in the intervening space.

From the particulars which have been brought forward—the replacement of the natural structure of the organ by contractile fibroid tissue—it might be presumed that the circulation through it would become greatly obstructed; and by experiment this is found to be the case. It is not necessary to repeat the details of experiments which have been already published, but it was found, by passing water through the blood-vessels of various natural and diseased kidneys, that with granular degeneration the kidney could not, on an average, transmit one-fourth as much water as passed through a healthy kidney under the same circumstances. The experiments were made by passing water with a fixed pressure and temperature into the renal artery, and measuring the amount which escaped by the vein in a certain time.[1]

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[1] See paper by Dr. Bristowe, Path. Trans., vol. ix., p. 309. Also Dr. Hughes Bennett, Clinical Medicine, 4th ed., p. 800. Also paper by author, Med.-Chir. Trans., 1860, p. 239.

Together with the renal disturbance other organs and structures become involved as the disease progresses in changes which are, for the most part, simple consequences of the original lesion. Some of them will be sufficiently referred to in the ensuing account of the symptoms and secondary affections belonging to the disorder; and in a chapter on the condition of the heart and arteries in chronic renal disease is a disquisition upon the nature and relations of the cardio-vascular change which is present with almost invariable regularity when the kidneys are advanced in the granular state, and is practically the most important of the pathological associates of the disease. The cerebral extravasation, the epistaxis, and the retinitis of granular degeneration are but results of this general condition. In the chapter referred to I have given reasons for believing that the cardio-vascular change is constantly produced by the renal; at the same time I think it must be admitted that a similar arterial and cardiac condition may be produced by other than renal causes; at least we can sometimes find evidence of it, while as yet there is no reason to suppose that the kidneys are otherwise than healthy.

The change—of which a more full account, together with several illustrations, is to be found in a later chapter—may be briefly described as one of thickening, together with degeneration of the muscular coat of the arteries, thickening of their fibroid sheath, and ventricular hypertrophy, these changes affecting mainly the systemic vessels, but occasionally traceable in the pulmonary. Not to reduplicate discussions which will be found elsewhere, it may be presumed that both the vascular and the cardiac change are due to a resistance probably in the capillaries to the transmission of the contaminated and unnatural blood. That the blood encounters abnormal resistance in its course is evident by the habitual fulness of the arterial system, from whence it is obvious that it cannot escape with its normal facility. This fulness or distention of the arteries is sensible to the finger in the characteristic tendon-like hardness of the pulse, and is capable of exact appreciation with instruments of the graphic and metric sort. This overfulness of the arteries would appear to be the cause both of the changes in their coats and of the ventricular hypertrophy. Heart and arteries exert themselves in succession, but not the less in unison, to drive the blood through the difficult exit of the arterial system, and both become hypertrophied in the effort.[2] The arteries, besides mere muscular thickening, undergo degenerative and pseudo-inflammatory changes, palpable atheroma, nuclear and muscular degeneration, and marked thickening of the fibroid sheath. With the thickening thus complex, there is not increase but diminution of elasticity and strength; the arteries become brittle. The ventricle, on the other hand, less liable to degenerative change, possibly because a smaller proportion of its. thickness is immediately exposed to the irritative effect of the abnormal blood, gains chiefly in pure hypertrophy and absolute power; and thus with increased force of heart and lessened strength of vessel occur the numerous extravasations and hemorrhages which characterize the disease.

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[1] Med.-Chir. Trans., 1860, p. 243.

[2] In addition to the sphygmograph Dr. Handfield Jones's sphygmometer is of especial use in determining arterial tension.

According to the views of Gull and Sutton these cardio-vascular changes are not consequent upon, but coeval with, the renal; the vascular system and the kidneys taking part simultaneously but independently in a deterioration common to the whole body, and allied to senile decay. I have elsewhere given the reasons which have led me to think that the old view is so far the true one, that renal disease, sole and unassisted, is a constant and efficient cause of the arterial change in question; but it may be that this state acknowledges also other sources; habitual impurity of blood due to other circumstances may possibly cause a similar series of changes; and I think it is impossible to note the effects of alcohol in brewers' men and the like without recognizing that they are prone to hypertrophy of the heart and vascular deterioration, unaccompanied by renal disease, or, should it exist, out of proportion to it. It is not my purpose here to consider arterial and cardiac thickening in all their relations, but only to insist upon their constant occurrence as direct and simple consequences of renal fibrosis. Whether the kidneys have become granular, as they most frequently do, as the result of gradual interstitial fibroid growth, or as the consequence of fibrosis secondary to an acute attack of renal inflammation, the result is the same. In the latter instance the cardio-vascular change may be apparent while yet the kidneys are smooth and unshrunk.

CHAPTER VIII.

CLINICAL HISTORY OF GRANULAR DEGENERATION.

It will be convenient, before proceeding to the symptoms and effects of granular degeneration, to consider its distribution between the sexes, the ages at which it is liable to occur, the conditions or morbid tendencies, if any exist, which predispose to it, and the circumstances, external to the body, or arising from within, which have the direct power of producing it.

Subjects.

The male sex is more liable than the female to granular degeneration of the kidneys. The difference is even more decided than is the case with nephritis. Taking 250 cases collected from the St. George's records, extending over a period of ten years, in each case this form of kidney being distinctly described as found at the *post-mortem* examination, 165 of the subjects were male, 85 female; the proportion being nearly 2 to 1. In 67 fatal cases under my own observation, in which the condition of the kidney was ascertained after death, 46 were male, 21 female, again a proportion of about 2 to 1. It will presently be shown that some of the causes of the disorder particularly affect the male sex. In a great number of cases the disease is associated with gout or with lead-poisoning. Gout seldom affects women, while, from the nature of their occupations, women are little exposed to the influence of lead. These circumstances may go a great way toward accounting for the unequal distribution of the disease between the sexes.

This disorder affects most the periods of life which are least amenable to tubal inflammation. It essentially belongs to advancing as the other to early years. But except perhaps the very earliest, no time of life is exempt from the possibility of its occurrence. The earliest instance of the disorder I am acquainted with was brought under my notice by Dr. Barlow of Manchester, who sent me a kidney in a typical state of granular contraction, which had been taken from the body of a girl only five years of age. A patient of the same sex died at the age of 10, under my late colleague Dr. Hillier, with a most marked condition of granular contraction affecting especially one kidney. A boy died at St. George's, under Dr. Ogle, at the age of 11, with granular degeneration traceable to scarlatina. A girl and a boy under my own care have been elsewhere referred to as having fallen victims to the disorder at the ages respectively of 12 and 14. And later the disease occurs with diminishing rarity as years advance, until between 20 and 30 it is found, not indeed often, but with a frequency which can be numerically expressed. After 30 and toward the approach of 40 it becomes more common, and at about 50 attains its greatest mortality. It, however, is very destructive for the whole time between 40 and 60, after which, though productive of a much smaller number of deaths, it continues to number its victims to the extreme limits of human life. The collection of cases already referred to from St. George's books contains two instances where this form of disease was described as occurring at the age of 82, and up to this age it must be regarded as frequent, allowing for the small number of persons left alive at this advanced period. The accompanying statement will show the ages at which the disease terminated in 242 instances collected from the *post-mortem* books:

Age in years.	Number fatal at stated period.
0 to 10	0
11 to 20	1
21 to 30	17
31 to 40	38
41 to 50	73
51 to 60	55
61 to 70	43
Over 70	15

CAUSES AND ANTECEDENTS.

Granular degeneration of the kidneys, unlike tubal inflammation, is necessarily a chronic disease. It has nothing of inflammatory haste. If the gradual changes in the fibrous tissue which constitute the disorder can be described as inflammatory, the inflammation is of such a slowly progressive sort that it is only in exceptional cases that it is possible to fix its commencement. Resulting from insidious changes in the anatomy of the organs which give no evidence of their presence until they have attained an extent which of itself is a record of prolonged morbid action, it is evident that the causes must be of a more remote and more protracted kind than those which have been shown as liable to set up the more acute disorder.

Examining the histories of patients who have been ascertained to have died of granular degeneration, we find they generally agree in two particulars: the appearance of the symptoms has been gradual; no obvious cause can be assigned. Here and there in large experience a case may be recognized as the sequel of scarlatinal or of some other form of nephritis; but in the overwhelming majority of instances the origin of the complaint must be sought, not in chance exposures nor in transient circumstances of any kind, but in influences of a continuous nature.

The question will first occur whether the disorder is in any way related to the tubercular diathesis, and this must be answered in the negative.

The obscure and gradual manner in which the disorder often arises leads one to search somewhat narrowly into any constitutional vice which may act as a latent source of organic change. In the series of 250 cases of granular degeneration already referred to, collected from ten years' *post-mortem* experience at St. George's, I found that tubercle existed in 20.8 per cent. Again, in 67 fatal cases seen by myself, and examined after death, there were 12 in whom tubercle was found in some part of the body; generally only a trace, often a mere tubercular cicatrix in the lung. From both these sources of information it appears that tubercle occurs in persons thus affected with less than ordinary frequency, estimating that one-fourth of those who die in London have tubercle in their bodies. It hence appears that patients who suffer from granular degeneration have had no proclivity to tubercular disease; in other words, that the chronic changes from which it arises are no part of the strumous diathesis.

The most marked predisposing cause of the disease is the climate of the temperate zone; or rather of such parts of it as approximate to the temperature of the British Isles. Further particulars bearing on this part of the subject will be found in the chapter on Climate.

Whether predisposing or exciting, hereditary influence is sometimes the sole recognizable cause of the disorder. The disease perhaps cleaves less to race than tubercle, cancer, stone, or diabetes; it is more often due to external and less often to inherent influences than are these affections; but yet occasionally the family proclivity declares itself unmistakably. I may refer to the medical history of an ancient house in which renal disease certainly of this nature has been transmitted from generation to generation, as if by an inexorable and unhappy entail. The details, as they relate to the last three generations, which were obtained through my friend Mr. Pollock, from a source which is beyond question, are as follows:

The first generation whereof the record treats consisted of a brother and four sisters. The brother died from an unknown cause at the age of 34, suddenly, but after long wasting. Two of the sisters died at the ages of 49 and 48 respectively, both having had albuminuria for many years.

The brother left two sons and four daughters. One of the sons died at the age of 26, having had albuminuria from the age of 12. Of the daughters three became the subjects of the same disease. One, Lady ———, died of it, with more or less œdema, at the age of 39, having had it since she was 16. Two others, still living, at the ages respectively of 38 and 40, are similarly affected, but it is not known at what date they became so.

The third generation consists of the six children of Lady ———, two sons and four daughters. All are alive, but five are the subjects of albuminuria. The first-born, a daughter, now 21 years of age, has had albuminuria from the age of nine months. The next, a son, now 20, has albuminous urine, but it is not known when it became so. The third appears to have escaped hitherto. The fourth, a son, now 16, has had albuminuria in an intermittent form from early boyhood. The fifth, a son, now 15, has had albuminuria in a marked form for two years. The sixth and last, a girl, now five years old, has passed urine which has contained decided but variable amounts of albumen from the age of six months.

It is possible that this remarkable chapter in the genealogy of disease might have been still longer had the condition been recognizable at an earlier date. A member of this family observes that portraits which exist as far back as the Wars of the Roses show that his ancestors were remarkable for a complexion of clear transparent pallor, such as was noticeable particularly in the case of Lady ———. This suggests that the disease may have been transmitted for longer than the three generations in which the state of medical knowledge enabled it to be recognized.

The following are the more obvious and immediate conditions to which granular degeneration of the kidneys can be traced:

1. The gouty habit from whatever circumstance it arise, but more especially when it is associated with lead.

2. Independently of gout, the presence of lead, alcohol, and possibly of some other irritants, in the circulation.

3. Conditions more especially of cardiac disease which produce and maintain venous congestion of the kidney.

4. Pregnancy, possibly acting by similar means.

5. Intermittent fever (?).

6. As one of the less frequent causes, obstruction to the exit of urine.

7. Prolonged mental disturbance, anxiety or grief. This cause of the disease is perhaps problematical; the mode of its operation is not obvious, but must be surmised as through the nervous system. A lowering of nervous force is to be recognized at least as predisposing to every form of albuminuria. I have seen so many instances in which granular degeneration has been immediately sequent upon trouble that in the absence of other causes I am fain to conclude that mental conditions are sometimes concerned in its production; and I am glad to hear from Dr. Clifford Allbutt, of Leeds, that this observer has been independently led to a similar inference; he indeed anticipated me in giving expression to it.

8. As a cause which has place—though there are differences of opinion as to whether it is of small or of paramount importance—a general fibrotic tendency affecting many organs and tissues, notably the arteries and the kidneys.

Finally, it must be allowed that the disease arises in certain cases in consequence of an organic tendency peculiar to the individual, or as the result of influences of which as yet we know nothing. It frequently comes on in a manner which, for want of more complete knowledge, we must call spontaneous. Persons with whom it is not hereditary, of temperate habits, who have been free from all the recognized antecedents of the disease, become subjects of it, we cannot tell why. Exposure to the climate of Great Britain, much as it may predispose to the disease, does not supply a reason why one Englishman should have it rather than another. That the renal alteration is not necessarily a part of a general degeneration is shown by the facts that the disease may arise from local disturbances of circulation, and that it sometimes occurs (in a comparatively rapid form, and in early life) without the concurrence of any similar change in other organs.

I will proceed to touch upon some of these causes of granular degeneration in further detail.

Gout, and Lead and Alcohol.

The disease is a frequent result or accompaniment of gout; this is by far the most important fact in its etiology. It is one of the results of the gouty diathesis, and may either precede or follow the external manifestations of the disease. The association of granular degeneration with gout is one of the most undoubted of pathological facts. Dr. Todd was, I believe, the first to draw attention to this relationship.[1] He published several cases in which gout was accompanied by albuminuria, which *post-mortem* examination showed to depend upon a granular, contracted, and cysted condition of the kidneys. Dr. Garrod has also dwelt upon this condition of the kidneys as connected with gout, and has given numerous cases.[2] He describes particularly a deposit of crystalline urate of soda in the tissue between the tubes, and states that he has only seen one instance in which the *post-mortem* examination of a gouty patient has failed to show distinct affection of these organs.

I found that among 69 cases of fatal granular degeneration there were 16 in whom the disorder was dependent upon or coincident with gout. It is scarcely necessary to insist that in such cases the gouty condition comes first; the renal mischief follows as a consequence. Disease of the kidneys does not appear to set up the constitutional disorder, for, however the kidneys may have been affected by disease other than granular degeneration, gout is not known to follow.

In the histories of cases where articular gout and albuminuria have coexisted the joint affection is usually the first to appear, and frequently dates back so far that, chronic as granular degeneration sometimes is, it is difficult to suppose that it could have anticipated the external symptoms. I knew a case, at last fatal from the renal disease, where gouty symptoms had existed, off and on, for twenty-six years ; and such instances are by no means uncommon.

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[1] Clinical Lectures by Dr. Todd, Urinary Diseases. Lecture 12.

[2] Garrod on Gout, 1859, p. 236.

The long precedence of the external symptoms of gout is particularly noticed when the disease is connected with hereditary influence or good living.

The gouty condition, as Dr. Garrod has shown, is one of the results of the absorption of lead. He states that about 30 per cent. of gouty patients in hospital practice have been under this influence. This accounts for the frequency of granular degeneration in plumbers, painters, compositors, and others who have been conversant with that metal. If a man who has a blue line on the gums have also albuminuria it is almost certain that he has also this variety of renal disease.

Looking back through the hospital records for a period of seven years, during which I was concerned in keeping them, I find that 42 workmen having to do with lead, as painters, plumbers, tin-workers, and compositors, died from disease or accident, and were examined in St. George's. This includes both surgical and medical cases. Of this number 26 had distinct granular degeneration of kidneys, in most of which that disease has led to the death of the patient. It is clear that the action of the lead is the cause, direct or indirect, of this astonishing proportion of renal disease. Whatever be the occupation of the person exposed to the influence of lead, whether painter, compositor, plumber, or tin-worker, he appears to have the same tendency to granular degeneration. The mischief is due to the metallic poison, which all share. Granular degeneration is, with few exceptions which take the shape of tubal nephritis, the only form of renal disease which lead appears to induce. Among the number stated there was but one instance of the occurrence of any other renal affection, and that was clearly due to cold and exposure.

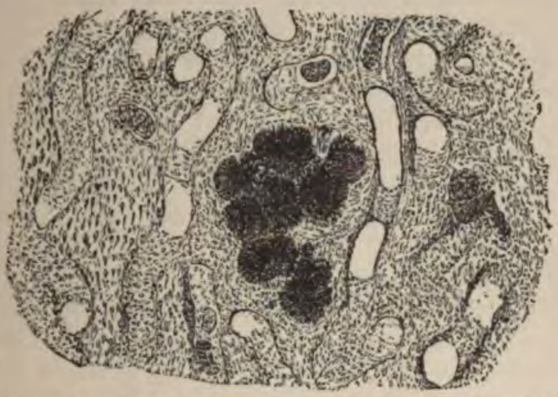
Constantly as this cause operates, the number of the laboring community who are exposed to saturnine influence is comparatively small, and it will be interesting to inquire what proportion lead-poisoning bears to other morbid influences as a cause of the disease. I have notes of 45 men who died of granular degeneration, of whom the occupation was known. Of these 10 had been exposed to this influence, 9 were painters ; 1 a compositor, with evidence of lead-poisoning.

From these particulars it is not too much to assert that of painters at least one-half eventually die of granular degeneration of the kidneys ; while as compared to other external circumstances the influence of lead is a more fertile source of this disease than any other with which we are acquainted.

It is certain that many cases of renal disease thus produced are associated with gout, and the urate of soda often exists in the kidney. At the same time granular degeneration frequently occurs apparently in consequence of the metallic poison in cases where there have been no external gouty symptoms. The gouty affection of the joints and granular degeneration are associated as springing from a common cause. If the morbid tendency affect the joints we have the ordinary symptoms of gout; if the kidney, those characteristic of granular degeneration. It appears that where the gouty condition has resulted from alcoholic liquors it tends chiefly to the joints; when from lead, to the kidneys. The rich man enjoys long life with gout in his extremities, the artisan perishes perhaps before his limbs are touched, from change of the same nature in the kidneys.

The change must be regarded as gout of the kidney.

Gout manifests itself not only by a deposition of urate of soda in the cavities of joints, but by peculiar changes in certain of the fibro-cartilaginous and fibrous structures. Fibrous tissue is a chosen seat of the morbid action. The fibrous structures and the cellular tissue around joints become incrusted and infiltrated with urate of soda, and are at the same time thickened and indurated as by chronic inflammation. The ligamentum patellæ, the tendo-achillis, and the tendons of muscles, have been found to be thus altered, and sometimes are infiltrated with the crystalline deposit, or contain it in the form of small white specks in their substance. From what has been said heretofore it will be seen that the affection of the kidney in these cases is analogous to the effects of gout elsewhere. We find, as Dr. Garrod has pointed out, and I can confirm, that there occurs a deposition of urate of soda between the tubes, connected, that is, with the intertubular fibrous tissue of the gland. This portion of the organ becomes thickened by a sort of chronic inflammation; it contracts and compresses the tubes, and the granular kidney results. This description holds good whether the gouty condition has resulted from intemperance, from the absorption of lead, or from any other circumstance.



Section through one of the cones of an advanced granular kidney from a gouty subject, showing the crystalline masses of urate of soda in the intertubular fibrous tissue.

Alcohol, another renal irritant, the action of which upon the kidney is more fully considered in a later chapter, may produce a result of the same sort. There is a large, smooth, somewhat congested kidney, partly tubal and partly fibrotic, which is begotten of beer upon the persons of draymen. And alcohol in other shapes has an influence in causing granular contraction of this organ by a process of chronic irritation such as makes the liver cirrhotic, although other organs are more amenable than is the kidney to this influence, and other causes touch the kidney more nearly than does this.

VALVULAR DISEASE OF THE HEART AS A CAUSE OF GRANULAR DEGENERATION.

Continued venous congestion of any organ produces, as a necessary consequence, certain changes in its intimate structure. Sir W. Jenner, in a paper[1] upon "Congestion of the Heart," lays it down as a pathological law that mechanically induced congestion of any organ produces induration of its substance; and assigns as the immediate cause of the induration the interstitial exudation of lymph, which may be converted into fibrous tissue. In several cases Sir W. Jenner traces the process from a mechanical obstruction in the heart or lungs to continued venous congestion of several organs, and finally to changes in their nutrition, from which they become hard and tough in texture, and increased in bulk. As to the kidneys they are described in the cases which are adduced as congested, hard, and often granular.

The truth of these observations cannot be doubted by any one who has had pathological experience. In the dead-house no sequence is of more constant occurrence. In a case of chronic disease of the heart, particularly of the mitral valve, the state of the kidneys may generally be predicted as in one or another stage of the same process. They will at any rate be hard, red, and full of blood, and the capsules will adhere more firmly than natural. Their bulk may be increased, the surface remaining smooth. In this case the congestion has lasted long enough to produce general increase in the fibrous tissue of the organ, but not long enough to allow of subsequent contraction. The same process continuing, the new material contracts, the surface becomes uneven and granular, and cysts are developed.

Kidneys thus affected have a general red color, by which, as well as by the smallness of the granulations, they may be distinguished from those which have become granular independently of a congestive origin. In their minute anatomy, also, there are some differences between the two sorts. The cardiac variety is early characterized by an irregular accumulation of epithelium in the tubes, which helps to give the gland its peculiar hardness. This epithelial luxuriance is to be observed before superficial granulation or any sign of fibrosis; and together with vascular injection may long constitute the only recognizable alteration, and the sole cause of the characteristic hardness of the organ. Chronic fibroid thickening, however, will become superadded in a considerable proportion of cases should the embarrassment of circulation last, and may proceed to marked granulation; though I must say that it has often happened to me, in instances where the peculiar hardness of the tissue has led me to expect some fibrotic change, to fail in discovering any trace of interstitial nucleation or other evidence of fibrosis.

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[1] Med.-Chir. Trans., vol. lxiii.

But that granulation ensues so frequently that cardiac disorder is to be classed among the causes of this affection is certain. When it has become established, the symptoms are often characteristic; but for long the renal disturbance may only be declared by intermittent albuminuria, possibly with hemorrhage, the constitutional disturbance being chiefly that which is directly due to the state of the heart. The heart has the lead, and usually keeps it; progressive and fatal as it is, it is apt to terminate while yet the renal disorder is in a comparatively early stage.

The following facts will show how often valvular disease of the heart is accompanied by this change in the kidneys. In the course of five years, as Curator of the Museum, I made *post-mortem* examination of 153 persons with valvular disease—29 of these had the kidneys hard, congested, and increased in bulk, but still smooth. The kidneys in 67 had granular surfaces and more or less contracted cortices.

These facts will be seen to correspond pretty nearly with some results arrived at by Dr. Barclay, in a paper upon "Valvular Diseases of the Heart."[1] In an analysis of 79 cases of valvular disease he gives 28 as having granular kidneys.

The proportion is nearly the same in both series of observations. The kidneys are granular in more than a third of the cases of valvular disease.

In the analysis of cases of granular degeneration, at page 187 of work on Diabetes, it is seen, looking at the concurrence of the two affections in another light, that the valves are diseased in 43 per cent. of the cases of granular degeneration. This depends upon a double relationship. Valvular obstruction produces alterations in the kidney, as has been shown. Granular degeneration, as will presently appear, often gives rise to atheroma, and sometimes to endocarditis, both of which tend to produce valvular disease. But while acknowledging that in some cases the valvular disease is the secondary affection, it is clear that more frequently it is the first departure from health. It is common to find the interior of the heart healthy in cases of granular degeneration, comparatively rare to find the kidneys healthy where death has resulted from disease of the heart.

The following case, one among many, is an instance of the development of renal disease in consequence of valvular obstruction:

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[1] Med.-Chir. Trans., vol. xxxi., p. 196.

Valvular Disease of the Heart, with consequent Disease of Kidneys—Dropsy Albuminuria—Pericarditis—Death—Post-mortem Examination.

Sarah Uridge, forty years of age, died in St. George's Hospital, where she had frequently been a patient. For the last four years of her life she had symptoms of heart disease, palpitation, dropsy, cough, often with blood-streaked expectoration, orthopnœa, blueness and turgidity of face. The physical signs were such as led to the inference that she had mitral disease, with much dilatation. There was a faint systolic murmur at the apex; there was increased præcordial dulness, and the sounds were loud. The urine was generally scanty, lithatic, and albuminous. A fortnight before her death an intense friction sound was heard all over the præcordium. With this she had much tumultuous action of the heart, dyspnœa, and rapidly increasing dropsy. The friction nearly ceased after four days, and blueness of the face increased, delirium came on at night, and the patient sank.

Post-mortem examination.—It is not necessary to describe the organs in detail. The body was œdematous, and the peritoneal and pleural cavities contained much fluid. The pericardium was adherent, the cavity being occupied by a layer of recent lymph, a quarter of an inch in thickness. The heart was of large size. The auricles, particularly the right, were much dilated. All the valves were diseased. The aortic and pulmonary valves were fringed with minute beady granulations. The tricuspid valve-flaps were thickened and rigid, and the orifice was narrowed so as only to admit two fingers. The mitral orifice was narrowed so that only the point of a finger could be passed into it. The anterior flap was occupied by a dense mass of calcareous matter.

The peritoneum contained a large quantity of purulent serum, but the membrane itself was not over-vascular. The liver and spleen were surrounded by old adhesions. The liver was somewhat hobnailed on the surface, and its section showed much nutmeggy congestion. The kidneys weighed together 12½ oz. The surface of the left kidney was granular, and it contained a number of large cysts. One part of the surface of the right kidney was granular, and it contained a number of cysts. A part of the surface of the right kidney was marked by deep cicatrices—the rest was smooth. The cortical tissue seemed to retain its natural proportion.

The sequence of the several organic alterations is here tolerably clear. The heart affection was of very old date and preceded all the other disturbances. The granular degeneration of the kidney arose gradually, in consequence of the congestion to which it was subjected. A similar change took place in the liver from the same cause, but to a smaller extent. The symptoms of the renal disease were much masked by the state of the heart, to which, as it appeared, the dropsy was chiefly due. The urine, however, was persistently albuminous, while the albuminuria of mere congestion is transitory. Generally where the renal mischief is of cardiac origin the urine is, as in this instance, scanty and lithatic. The lithatic condition may be roughly taken as a warrant that the kidneys, however congested, have not reached the extreme of degeneration. Pale urine, with cardiac-renal disease, is of worse omen so far as the kidneys are concerned. The pericarditis, to which the death of the patient was chiefly to be attributed, was probably connected with the state of the kidneys rather than with the primary valvular lesion. This case is one of a sort which has importance from the frequency of the series of changes of which it is an example. As in this instance, it is usual for the symptoms during life to be conspicuously of cardiac origin. When granular degeneration occurs as a consequence of gout, a disease in itself nearly free from danger, it has time to progress and develop to the utmost extent consistent with life. But when it has sprung from cardiac obstruction the heart is the seat of mischief, which progresses at no slow rate, and which has necessarily attained a considerable power for evil before it can initiate the renal disease. The heart, therefore, usually directs the symptoms. But it is probable that the renal disease modifies their course, and it is the chief cause of the tendency to pericarditis which is so fatal in the circumstances.

Pregnancy as a Cause of Renal Disease.

Another condition—acting probably mechanically, and producing, as does heart disease, venous congestion of the kidney and the train of organic changes and constitutional results which spring therefrom—is pregnancy.

It has long been known that in a recognizable minority of cases the urine in the later months of pregnancy is albuminous, and that this condition entails a liability to convulsions resembling those of uræmia, which are apt to come on during labor or before. It was long ago asserted by the late Dr. Lever that puerperal convulsions are always preceded by albuminuria, and that they are essentially uræmic in their nature was afterward insisted on by Sir James Y. Simpson,[1] and is now, I believe, almost universally admitted, at least, as a general truth, if not as one without exception. That during pregnancy the urine may be albuminous and yet no convulsion follow is of common experience. But puerperal convulsions without renal disturbance appear to be, to say the least, of extreme infrequency. Blood drawn under attacks of this nature has been ascertained to contain a great excess of urea—1 in 960, or six times the normal amount, was found in one instance.[2] And the uræmic origin is further borne out by the symptoms of the seizure, the increased vascular tension with which it is accompanied, and its susceptibility of relief from bloodletting and depletion.

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[1] Edin. Med. Journal, October, 1852.

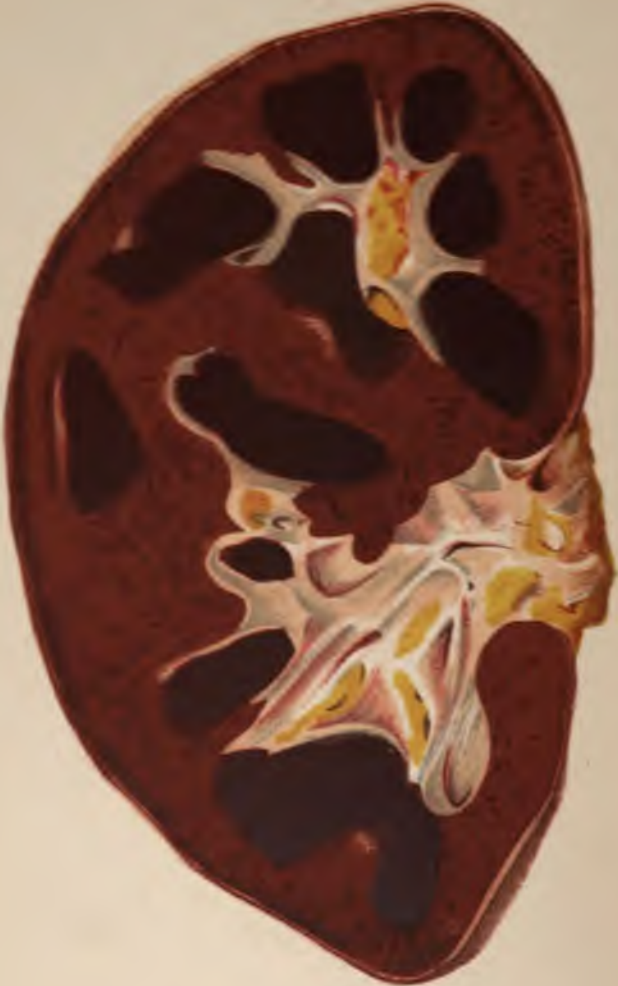
[2] Puerperal Diseases, by Dr. Fordyce Barker, p. 100. London, 1874.

Such attacks, however, are only some of the symptoms which accompany the albuminuria of pregnancy. The legs are generally œdematous—a symptom which is not always renal, since it is sometimes produced apparently by pressure on the ascending cava, while the urine remains free from albumen. The face is sometimes œdematous, and disturbances of vision occur connected, as it appears, with more or less albuminuric retinitis.

Dr. Lever believed that the albuminuria of pregnancy was due to the pressure exerted by the gravid uterus upon the kidneys and their blood-vessels, and their consequent congestion and embarrassment. And all the circumstances of the affection appear to corroborate this view. Both convulsions and albuminuria are most frequent in first pregnancies, where the structures are less yielding and the pressure greater than afterward. Dr. Litzman found the urine to be albuminous in 37 of 131 pregnant or lately delivered females; of the 37, 26 were *primiparæ.* Dr. Blot detected the same state in 41 pregnant women out of 205, most of those in whom it was found being in their first pregnancy. And passing to the revelations of morbid anatomy, the condition of kidney, whether temporarily embarrassed or as the result of frequent repetition of the process, permanently diseased, is one which obstructive or venous congestion would be apt to produce. The changes in this organ are nearly allied to those which occur as the result of heart disease with attendant venous repletion. They may be epitomized as congestion succeeded by excessive growth of epithelium, interstitial nucleation, fibrosis, and granulation. But when of uterine origin it is to be observed that, partly perhaps by reason of the susceptibility of the subjects, it is more mischievous than when its source is cardiac. And other differences can be pointed out, notwithstanding the essential similarity between the uterine and the cardiac kidney. Both undergo increase, at first of epithelium and ultimately of fibrous tissue; both become at last granular; but the cardiac kidney is usually to the last red, hard, and free from oil, while that of uterine origin often becomes fawn-colored and fatty.

It has been supposed by some writers that the necessity of getting rid of some excrementitious product of gestation is the chief cause of the renal embarrassment; but such a surmise would seem somewhat gratuitous in view of the congestive nature of the renal lesion and the possible cause for venous obstruction as regards this organ in compression by the gravid uterus of the emulgent veins. The date at which the symptoms usually begin—after the uterus has left the pelvic and entered the abdominal cavity—and their increase with its increasing bulk; the greater frequency of the affection when the structures have not been made distensible by antecedent pregnancies; and these, together with the evidence of venous obstruction which often accompanies pregnancy, in swelling of the lower limbs, inde-

Plate 6



KIDNEY OF PUERPERAL CONVULSIONS. *[From case of S. Van G. Page 161]*

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PLATE VI., *to face page* 117.

Section of the Kidney of a woman who died of Puerperal Convulsions, having had Albuminuria and Œdema during her pregnancy. The case is given at length, page 117. (S. Van G.)

The section shows a state of extreme congestion without any of that white opacity which is evidence of a deposit in the tubes. The cones are of a deep purple color, the cortex rather more florid. The gland is considerably increased in bulk, chiefly in consequence of the injection of the vessels. The surface is not shown, but was perfectly smooth and of a similar color to the section.

pendently of albuminuria, concur to assign the result mainly at least to mechanical causes.

The disproportionate results, as compared with other forms of renal disease, particularly in respect of convulsions, which have been stated to occur in one-fourth of the cases of the albuminuria of pregnancy, may be perhaps due to the disturbing influence of labor, the exaltation of nervous susceptibility which would seem to accompany the act, or the effect which it has, whether by exhaustion or febrile action, of destroying the equilibrium of the nervous system. Thus, under the sudden influence of parturition, uræmic convulsions may result from an amount of uræmia which might otherwise be inadequate to produce them.

Usually with the emptying of the uterus the kidney will right itself and resume its former condition, but now and then, especially if pregnancy be often and quickly repeated, the state will recur with each, until finally the albuminuria becomes permanent and the granular kidney is irretrievably produced.

I will relate some instances of the disease which will help to supply some necessary details.

In the following case the condition of kidney attained in a first pregnancy, the first stage of the disease, is shown.

Puerperal Convulsions after a first Labor—Urine Albuminous—Death—Post-mortem Examination.

Sarah Van G., twenty-one years old, unmarried, died at Queen Charlotte's Hospital, under the care of Dr. Brodie, who kindly furnished the following particulars, and gave me an opportunity of examining the body.

She became pregnant, and was delivered in the hospital of a first child, after a perfectly natural labor. She had always had good health previously, excepting that the legs and feet had been œdematous during the later months of her pregnancy. After the child was born she passed a comfortable night, and seemed to be going on well for sixteen hours, when she had a convulsive fit which lasted ten minutes. In about an hour she had another, which was of longer duration and more severe; the face became more congested, and it was longer before consciousness returned. In a third fit, which occurred after an interval of two hours, she died.

Some urine which had been obtained was amber-colored, acid, clear and albuminous (albumen = $\frac{1}{6}$).

There was a little œdema about the lower extremities. The brain was natural in all respects, the ventricles empty. The lungs were slightly congested. There were old adhesions in the pleuræ and pericardium. The aortic and mitral valves were thickened to a trifling degree by old deposit. The liver was large and slightly fatty; there were adhesions connecting it with the diaphragm. The uterus was firmly contracted, about the size of a cocoanut, and natural in all respects.

The kidneys are accurately represented in the accompanying drawing. They were of about the natural size, but had the appearance of being swollen, looking round and full, one weighing 4¾ oz. The capsules were adherent and slightly thickened. The surfaces were smooth, and intensely injected; no stellate vessels were visible, but the injection was minute and uniform. On section the pelves were seen to contain a little turbid urine, but were not dilated. The mucous membrane was somewhat injected. The renal substance was intensely injected throughout; the cones a deep purple color; the cortex of a lighter tint. The latter had nearly lost the faintly linear arrangement proper to it, and had an uniform structure like that of close red sandstone, but of a brighter red color. It was harder and denser than natural, and was relatively increased in bulk.

A careful microscopic examination was made both of the fresh tissue and after various methods of hardening. The epithelium was natural, but was accumulated in the cortical tubes to the general opacity of the section, but not so as to cause decided distention. The malpighian bodies were fully injected, the fibrous matrix conspicuous, and the nuclei pervading it abnormally multiplied.

This case is brought forward for the sake of the morbid anatomy; the clinical facts are, unfortunately, sufficiently familiar. The condition of the kidney is undoubtedly one which often leads to granular contraction, and the interstitial hypernucleation shows the beginning of the fibrotic process. The state, both in the epithelial excess and in the interstitial change, is closely analogous to that which results from passive congestion from other causes.

The change in the kidneys, as found after death from puerperal convulsions, seems too small to account for the fatal result. But it is to be considered that it is not the kidney alone which occasions the mischief. The patient may be regarded as one suffering from a certain amount of uræmia —an amount of uræmia probably harmless so long as surrounding circumstances go smoothly—who is suddenly made preternaturally impressible by loss of blood and nervous exhaustion. The susceptibility of the nervous centres thus suddenly increased, uræmic convulsions result from an apparently inadequate amount of renal disease.

In two other instances in which I was able to examine the kidneys in a like early stage of the disease after death by puerperal convulsions I could find no other alteration save congestion and universal and uniform epithelial accumulation. In one of them a general yellowish tint of cortex showed through the prevailing hyperæmia, and a small proportion of the epithelial cells were fatty, the rest being absolutely natural. In these it is possible that had the patients survived the immediate results of the conjunction of labor with albuminuria, the kidneys might have recovered without any tendency to granulate.

A case formerly under my care, which has recently terminated under that of a colleague, exemplifies a later phase of the disorder.

A young woman who had been once released from the dangers of matrimony by the death of her husband, but unfortunately exposed herself to them a second time, had convulsions after her third labor, and was brought to the hospital with severe general dropsy, attended with albuminous urine, dimness of sight, and the retinal changes characteristic of renal disease. She was dismissed in apparent health, but still with albuminous urine, and a hint that another pregnancy would bring further peril. A year later she was brought back in the course of a series of convulsions of uræmic type, nearly suppressed albuminous urine, and the tumor of early pregnancy in the abdomen. She aborted and died. The kidneys were granular externally. They were of pale color and fatty texture, and increased in size to the conjoint weight of 15 ounces. The liver was fatty and weighed 69 ounces. The heart was somewhat hypertrophied ; it weighed 12 ounces ; the mitral valve was slightly thickened.

The chance of progressive disease resulting is of course increased by frequent recurrence of pregnancy. The following case appears to be an example of granular degeneration gaining ground with successive repetitions of the cause :

Amelia Teal, thirty years of age, a native of Germany, came under notice while pregnant for the sixth time. Every pregnancy had been attended with swelling of the legs. She was admitted into St. George's Hospital under the care of Dr. Bence Jones, in November, 1856, with general œdema and albuminous urine. She was at that time in the second month. As the pregnancy advanced the urine became more albuminous. It was at first clear, but afterward became smoky from the admixture of blood. Casts were found, at first transparent, latterly containing cells of renal epithelium or granular matter. They were always of medium diameter. By April, being then in the seventh month, the albumen had increased so that the coagulum occupied three-fourths of the tube. The urine was diminished in quantity ; sp. gr. 1015. It was faintly acid. The œdema had now become considerable in the lower extremities, and the face was puffy and pale. There was constant pain in the loins and frequent vomiting. There was much dyspnœa, so that the patient was obliged to sit upright. Toward the end of the month labor came on, and she sank, apparently from exhaustion, two days afterward.

At the *post-mortem* examination there was considerable anasarca. The state of the uterine organs was such as is generally found in such circumstances. The heart and lungs were natural, but there was a good deal of serous fluid in the pleuræ and pericardium. The liver was rather fatty. The other organs, excepting the kidneys, were natural.

The kidneys were considerably increased in size. The capsules were slightly adherent in one or two spots, but generally came off easily, leaving a surface nearly smooth to the touch, but having an appearance of incomplete large granulation. The general color was a sort of whity brown, something like that of oatmeal porridge. The lobular markings on the surface had disappeared. On section the cones and cortex maintained about their natural proportion to each other, and both had much the same color as was presented by the outside. Around the outer edge of each cone was a halo of fine radiating lines of a yellow color. There were no cysts.

When a hardened section was examined with a microscope there was found to be a considerable formation of new fibroid tissue as a layer spread beneath the capsule. In this could be seen the remains of tubes in a contracted or compressed state, separated from each other by the new growth. The new tissue was spread more evenly than usual, not being divided into processes. The capsules of the malpighian bodies were generally thickened. The tubes near the central parts of the gland were variously dilated.

By examining in the fresh state it was found that the convoluted tubes were more or less choked up with finely divided oil, particularly where the yellow radiating lines were observed. The tubes so affected were generally about $\frac{1}{400}$ of an inch in diameter.

Globules of oil were seen upon the malpighian bodies. The straight tubes were variously packed with epithelial cells, or with amorphous granular matter. Their contents were such as had formed the casts.

The epithelium was for the most part natural, but a certain proportion of it was fatty.

In this case the constant recurrence of œdema with each successive pregnancy, and the final appearance of decided renal disease with the last, is strong evidence that the disease was set up by the state of the uterus.

The condition of the kidneys was that of an early stage of granular degeneration. There was abundant evidence of an intertubular formation, though as yet contraction had not proceeded to the extent of distinct unevenness of surface. As appears to be often the case when this disease results from pregnancy, it was accompanied with fatty degeneration.

Dr. Braun describes with minuteness the state of the kidneys in women who have died of puerperal convulsions. His experience is based on the results of twelve *post-mortem* examinations. He describes the organs as being in one of three conditions.

The first condition is that of extreme congestion. The superficial vessels are dilated and full of dark blood, the cortical substance is brownish red, soft and friable ; from the surface of a section there flows sticky bloody fluid, with which the parenchyma is infiltrated. The cones are hyperæmic, and also the mucous membrane of the pelves. Hemorrhagic effusions are sometimes seen.

In the second stage the congestion has given place to a general dull yellow color. The kidneys are larger than natural. "The surface is sometimes smooth, sometimes granulated, covered with elevations of the size of a poppy-seed." There is more or less fatty degeneration of the epithelium on the malpighian bodies.

In the third stage the kidneys are reduced to their normal dimensions, or even sink below them. The capsule is thickened and adherent, the surface of the kidney is uneven, tuberculated, and often shows deep furrow-like indentations dividing it into lobes. The cortical substance has wasted, and the organ is generally tough in texture.[1]

Such, according to Dr. Braun, are the conditions of the kidneys after death by puerperal convulsions; the last, more advanced, stage being less frequent than the other two.

Thus, the results of his large experience accord with the testimony which has been already adduced in regarding the "puerperal kidney" as one of intense venous congestion, such as mechanical causes would produce, succeeded by incomplete or well-marked granular degeneration. It would be easy, if it were necessary, to collect from clinical records numerous instances in which granular degeneration of the kidneys has succeeded upon the changes induced by pregnancy. The following may be adduced as examples, though the sequence will probably be regarded as placed beyond doubt by the evidence which has been already brought forward.

Professor Simpson relates a fatal case of puerperal convulsions during a third pregnancy; the attacks had come on, as was supposed, in consequence of mental excitement. "The kidneys presented a well-marked specimen of granular degeneration, probably of some standing."[2]

Dr. Roberts, in his valuable work on Renal Diseases, gives two cases in which the kidneys have become diseased in consequence of pregnancy. They are shortly as follows:

A married woman, thirty-nine years of age, while in the third month of her sixth pregnancy, had frequency of micturition, œdema of the face and legs, and albuminous urine. She miscarried at the fifth month, but the urine continued albuminous. Transparent casts, and others containing epithelium, were found. The dropsy disappeared, but the patient had repeated attacks of convulsion, and more than a year after the symptoms had commenced died in a state of coma. After death the kidneys were found to be granular and atrophied.

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[1] Dr. Braun, on the Anæmic Convulsions of Pregnancy, Parturition, and Childbed. Translated by Dr. Duncan, 1857.

[2] Edinburgh Monthly Journal, October, 1852. (Case 1.)

In this case it may be presumed that the renal mischief had begun in some of the preceding pregnancies.

Another case, quoted by the same author, is that of a servant-girl who became pregnant at the age of 26. Toward the end of her time the legs became œdematous. The dropsy disappeared after the birth of the child, and for two years she remained in apparent health. General, but not excessive, œdema then came on, conjoined with fugitive disorder of vision, occasional attacks of diarrhœa, and general failure of health. When seen medically the urine was found to be pale and albuminous, and the dropsy had extended to one pleural cavity. The sight afterward became more impaired, and the patient died comatose after a series of convulsive attacks. Death took place about three years after the first appearance of dropsy. "The kidneys were in a state of fatty degeneration, with beginning granular atrophy."

This case is valuable as taken in conjunction with the others. It is probable that the renal mischief was started by the pregnancy, and gave rise to the œdema which was then observed; the changes progressed afterward in the latent manner in which granular degeneration proceeds, and terminated in the well-marked disease which caused the death of the patient.

To touch upon clinical apart from pathological experience, I have seen a considerable number of instances of persistent albuminuria with characteristic symptoms, general, and in one case laryngeal, œdema, which had begun in a first and become exasperated by subsequent pregnancies. Noteworthy points about them are, first the marked increase of the disorder, and the increasing risk of convulsion, which each successive gestation involves; and the slight tendency to get worse, or even the occurrence of steady though slow improvement both in the general symptoms and in the state of the urine, so long as the condition in which the disorder has arisen be not repeated. A practical rule is to be discerned which it is not always easy to enforce.

SCARLATINA.

Among the causes of this disease Scarlatina must take a place. It is probable that acute renal inflammation, whatever its origin, may, however infrequently, eventuate in granulation. Scarlatinal nephritis, signalized by dropsy, may be followed or attended by interstitial inflammation, and that by fibrosis, contraction, and granulation. Of this pathological series examples have been already given.[1] But there is another and a less obvious process by which scarlatina may lead to this renal transformation. A child who has hitherto had perfect health takes scarlatina. He recovers without dropsy or ostensible renal mischief. But he does not regain his health. He remains languid, ailing, and pale, and perhaps becomes stunted in growth. After the lapse of years he is found to be the subject of chronic albuminuria, with light and pale urine, hypertrophy of the heart, and possibly retinal hemorrhage. He has, in fact, the granular kidney, which has insidiously begun in some undeclared inflammatory result of the treacherous and far-reaching exanthem. This appears to be an exceedingly infrequent mode of origin.

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[1] Page 65 et seq.

Intermittent Fever.

A cause of the disorder which is of infrequent occurrence, and must be spoken of somewhat doubtfully, is the influence of intermittent fever, or rather of that form of it which belongs to tropical climates. I have seen more than one instance of persons who have returned from India with persistent albuminuria and symptoms of the granular kidney after having while there suffered severely from malarious fever. Possibly the repeated internal congestion of the cold fit, to which the enlargement of the spleen bears witness, may be enough to initiate the fibrous hyperplasia, as other conditions of passive congestion have been shown to do, and advance it a step with each exacerbation until the organ is permanently injured. A gentleman in whom the disease had presumably this origin was in England and under my observation for 16 years. The urine was always pale, albuminous, and of low specific gravity, and the circumstances such as to place the organic condition beyond doubt. The disorder long remained without apparent progress, but at last the patient became the subject of uræmic asthma, and attacks of dyspnœa, together with convulsions of the same origin, concluded his long struggle against hopeless disease.

Retention of Urine as a Cause of Granular Degeneration.

Cases sometimes happen which give reason to suspect that the kidneys have become the subjects of granular change in consequence of the prolonged influence of accumulated urine. In young persons, with whom this form of renal disease is so rare that its absence may be generally reckoned upon, I have noticed it more than once in association with a history of stone or dilatation of pelvis or ureter, the result of former obstruction. I may instance the case of Dodd.[1] He was operated upon for stone at the age of 3. At the age of 14 he died of granular degeneration affecting both kidneys; one was in addition dilated and attenuated. Tillet, again, who died of the same disease at the age of 12, presented a somewhat similar condition. One kidney was much atrophied, the other slightly so; the pelvis of the more atrophied kidney and the ureter of the less atrophied were dilated; a past obstruction was necessarily inferred, but there was no clew as to its nature. And I could adduce other instances not numerously but sufficiently distinct in their results in which the same cause has led to the same result. More or less fibrosis of the kidney, together with glandular atrophy, is indeed not seldom to be recognized as a consequence of chronic retention of urine.

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[1] See work on Diabetes, p. 197.

Retained urine sets up, by pressure or contact, renal changes which are considered elsewhere. Some are allied to pyæmia and are attended with abscesses in the intertubal districts of the organ. And it is not impossible that lesser degrees of inflammation may be engendered about the blood-vessels by degrees of morbid absorption or other irritation insufficient to produce suppuration.

A measure of perivascular or interstitial inflammation of wide extent but low degree might conceivably originate the series of changes which culminate in the granular kidney. It is to be noted that the renal often appears to be connected with the obstructive lesion only as a remote or indirect sequence; the obstruction has long ceased to exist when the condition of kidney becomes manifest. As a consequence of present stricture, granulation or interstitial fibrosis is seldom to be observed.

General Fibrosis as a Cause of Renal Disease.

The relation of granular degeneration of the kidney to a general fibrotic change in which the arteries participate has been discussed elsewhere; it only remains to add a word or two touching the occasional participation of other viscera in a process such as that to which the kidneys have succumbed; though it would seem more correct to say that other organs are affected by causes such as tell upon the kidneys, than to talk in general terms of a fibrotic tendency.

It is known that one of the tendencies of old age is to fibroid thickening, and increase of fibrous tissue in many parts of the body. A less general fibroid degeneration results from the excessive use of alcoholic liquors, particularly ardent spirits. As the result of spirit-drinking the most marked effect is upon the liver and lungs, though the kidneys are not exempt. The influence which alcohol has in causing renal disease is considered elsewhere.

This general tendency, from whatever cause it arise, may affect the kidney in common with other organs. As a means of estimating the frequency with which granular degeneration occurs as part of a general fibroid change, I ascertained the proportion of cirrhosis of the liver and thickening of the capsule of the spleen as associated with granular kidneys. In 250 cases of granular degeneration the liver was cirrhosed in 37, a proportion of about

1 case in 7, while there was noticeable thickness or opacity of the capsule of the spleen in 47, a proportion approaching 1 in 5. These numbers may give a rough estimate of the frequency with which the exaggeration of fibrous tissue which constitutes granular degeneration has affected other organs besides the kidney. The proportion is not large. In this country the kidneys are prone to morbid actions, especially to such as affect their fibrous element. They suffer from influences which are concentrated upon themselves, while at the same time they are prone to participate in such general disturbances as promote the encroachment of fibrous tissue upon other organs. Their sympathy in this respect is most evident when the source of the change is valvular disease. When venous obstruction has resulted from uterine enlargement, the kidneys are usually affected alone. The hepatic and splenic veins are, from their position, free from the injurious pressure which the gravid uterus exerts upon the vessels which return the blood from the kidneys. Gout, too, attacks the kidneys while other viscera are exempt from its influence, though in this case the reason of the preference is unexplained.

In fine, it may be stated as a general law that chronic congestion, be it of what sort it may, so it be continued long enough, leads to granulation, which is the renal expression of fibroid hyperplasia. Thus, whether the congestion is passive, as the result of cardiac disease, uterine pressure, or aguish influx; or whether it is active, as due to the irritation of alcohol, lead, or the poison of gout, or the habitual effect of cutaneous compensation under a chilly climate and changeful sky, or kept up by the protraction of tubal inflammation, all the sources of the disease have this in common, that they are causes of long-continued or oft-repeated hyperæmia, perhaps merely mechanical, or connected possibly with functional stimulation.

CHAPTER IX.

SYMPTOMS OF GRANULAR DEGENERATION OF THE KIDNEY.

This disease has been described as the result of changes, at first insignificant, beginning in a certain part of the gland, and then creeping on, step by step, until decided alterations are produced in its construction. The symptoms are developed in the same insidious manner. It is impossible to recognize the disorder until it has reached what is really an advanced stage. The symptoms which then declare its existence are often of such a kind as to be easily attributed to diseases of other organs. A patient who has reached adult life may come under observation, suffering from dyspepsia and vomiting, or indefinitely out of health, with headache as the only definite symptom, or complaining only of depression of spirits, or with dimness of vision, or with bronchitis, or with slight and transient œdema, or with decided dropsy. With one or more of these symptoms it is noticed that he has an unhealthy look—somewhat of the "pallor luteus." He has perhaps somewhat puffy features, and somewhat of an anæmic appearance; though a sort of sunburnt tinge upon the skin prevents the whiteness characteristic of the more acute disease, and gives a sort of whity-brown hue to the face. The tendency to anæmia is less marked than with other forms of renal disease, and a blotchy or red-mottled complexion, or even decided "acne rosacea" is to be recognized as one of its signs.

Perhaps, after various measures have been unsuccessfully directed to some troublesome symptom, its renal origin is suspected, and albumen discovered in the urine. If now the patient be questioned as to his previous health, it is found that for some time, perhaps for years, it has been more or less broken. The urine has been increased in quantity, and passed more often than formerly. Perhaps it may have been noticed, on one or more occasions, that it was black with blood. If there should be any œdema it has come on gradually, without any such obvious cause as exposure, scarlatina, or intoxication.

This disease differs from other renal disorders in the obscurity of its commencement, and in the consequent difficulty in fixing its duration. While other renal affections are generally evident on their first appearance, and run a tolerably rapid course to recovery or death, it is scarcely possible with granular degeneration to say how long it may last. Essentially a chronic disease, it would be easy to multiply instances where it has been known to exist for ten, fifteen, or even twenty years. It happens, in the greater number of cases which end fatally in hospitals, that the symptoms can only be traced back for a comparatively short time, but this arises from the indifference of the working class to slight ailments; so that they only come under notice when the later symptoms of the disease have accumulated upon them, and they are incapacitated by dropsy or some of the consequences of uræmic poisoning.

One of the earliest symptoms which may lead to a suspicion of the disease is an increase in the quantity of urine, which is pale and of low specific gravity. It is passed more often than natural, especially at night, apparently in consequence of its increased quantity, not because it has acquired any irritating quality. If examined in this early stage it may be found perfectly free from albumen, or may contain only a minute trace, or a trace only after food or on getting up in the morning. Casts are sometimes to be found before the albumen is appreciable. So long as the urine retains its superabundance the patient will remain without obvious dropsy, though even then traces of it on the tibiæ or elsewhere may declare themselves to the medical observer. The earliest constitutional symptoms are often such as point to the stomach; dyspepsia is seldom absent, and sometimes the chief complaint. Diarrhœa, on the other hand, is exceptional. After a time the urine becomes decidedly, though not very highly, albuminous, and the complexion, especially if the urine be very pale, becomes discolored, as if some of the pigment which should have tinted that secretion had been used for the face. A shade even approaching that of gingerbread is sometimes to be recognized as a sign of advanced disease.

With the progress of the disorder vomiting may become frequent and distressing; it is more particularly to be noted in the morning before food; it is attended with much retching and the laborious produce of small quantities of white frothy or slimy matter, which often has an alkaline reaction and may give evidence of ammonia. The vomiting and nausea may be habitual, or be repeated chiefly in paroxysms lasting with violence for some hours or days, to pass off for a time almost completely. In a case in which the gastric symptoms were exceptionally prominent the loathing of food was such that vomiting was excited even by the distant sound of the dinner-bell; the intestines were ulcerated—a rare but not unknown complication of this form of renal disease. Vomiting is of evil import, as a frequent precursor of convulsion. Where stomach symptoms prevail head symptoms commonly conclude. And it must be noticed, too, that the retinal and other less direct results of uræmia are apt to be associated with its gastric manifestation.

The retinal mischief leading to various degrees of mistiness of sight, or partial or complete amaurosis, is but part of a change common to the arterial half of the circulating system, which, if the renal disease be advanced, is sure to show itself in other ways; tangibly in the hardness of the pulse, which remains permanently full like a subcutaneous cord; and no less surely in the hypertrophy of the left ventricle, a condition which seldom declares itself by any direct symptoms.

Pain in the loins occurs, but not constantly. It is occasionally somewhat severe, unaffected by flexure of the body, but aggravated by jolting or rough travelling; due, in such cases, as I have found, to a more than ordinary thickening of the capsule, as if by a low degree of perinephritis.

Dropsy is a symptom which, though far less constant than in other renal disorders, deserves a prominent mention in connection with granular degeneration. When considerable it marks an advanced stage of the disease, at which the previously copious urine has become scanty. Many persons go through the course of the disease without any dropsical effusion. 19 out of 68 patients (whose symptoms are detailed in the table on opposite page) went to their graves with granular degeneration, without dropsy at any period of the complaint. The first to occur is œdema; and it may be stated as a general rule, that if no œdema exist there is no effusion in the serous cavities. Next to the cellular tissue, the pleural cavities are the most often affected, then the peritoneum; the pericardium rarely. The dropsy is most conspicuous when the renal affection is conjoined with valvular disease of the heart. In such it is often difficult to say whether the heart or the kidney deserves most blame. Under these circumstances, the œdema often becomes so great, that were the case uncomplicated, it might be put down as one of tubal inflammation.

I have known, and in a case where there was little general swelling, an extraordinary amount of subconjunctival œdema, such as to overhang the irides and prevent the closure of the lids. It is probable that this local dropsy partakes, like the laryngeal œdema, of the nature of inflammation. In the instance referred to the conjunctivæ were much injected.

With the symptoms of granular degeneration must be mentioned, as among the less common, mental depression, sometimes with a lachrymose habit. This may occur in persons who are without any of the more obvious signs of the complaint, and be the first means of drawing attention to the state of the urine.

Attacks of dyspnœa, asthmatic in their type, are among the more distressing, though happily the less common, results of the disease. Agonizing difficulty of breathing, attended with much terror and apprehension on the part of the patient, who cannot move from the upright posture, is apt to come on suddenly at night, much after the manner of a paroxysm of asthma, though in a person not hitherto asthmatic. After a time it yields to copious frothy expectoration, which in one instance within my practice was often tinged with blood, and the lungs are found to have become suddenly pervaded with bronchial and moist sounds. These in their turn quickly subside, and in the morning, both in act and sound, the breathing may be perfectly natural. Such a seizure looks like an eliminative effort on the part of the bronchial tubes ; I have only seen it with advanced disease, hypertrophy of the heart, increased tension and evidence of uræmia. It often seems to have with it an element of cardiac distress ; and indeed in some cases seizures occur which appear mainly or wholly connected with ventricular hypertrophy of renal origin. Of such, instances are recorded at the conclusion of this chapter.

This review of the symptoms of the disease would be insufficient were it not to comprise a somewhat more particular account of some of the morbid conditions with which it is associated. Before proceeding, therefore, to the uræmic ending I shall briefly revert to some of its results and complications.

Analysis of 68 *Cases of Granular Degeneration under the Observation of the Author, and attested by Post-mortem Examination.*

Causes, showing number attributed to each.		Consequent Affections, how often present.		
Valvular disease of heart	6	Hæmaturia		10
Pregnancy	1	Frequency of micturition		13
Gout	16*	Pain in loins		9
Occurring in printers or compositors	10	Œdema		49
		Ascites		18
		Hydrothorax		23
		Fluid in pericardium		3
		Purpura		0
		Erysipelas or inflammation of cellular tissue		3
		Uræmic convulsions		11
		Simple coma. or semi-coma †		14
		Other head symptoms (neither fits nor coma) ‡		13
		Pneumonia		7
		Pleurisy	Present in a recent state at time of death.	7
		Peritonitis		3
		Pericarditis		16
		Endocarditis		4
		Bronchitis		24
		Vomiting (not bloody)		17
		Diarrhœa		2
		Congestion, or inflammation of bowels		3
		Amaurosis, or dimness of vision		5
		Sanguineous apoplexy		3
		Epistaxis		4
		Vomiting of blood		3
		Hypertrophy of heart, without valvular disease or pericarditis		31
		Atheroma		14

* Three of which associated with lead. † No convulsions occurring in these cases.
‡ Cases excluded where the symptoms of valvular disease have predominated.

CARDIAC AND ARTERIAL CHANGES.

One of the most frequent consequences of granular degeneration is hypertrophy of the left ventricle of the heart. In the 68 cases of which the details are annexed in the table, were 31 in which this change was sufficiently marked to call for notice at the *post-mortem*. This statement is exclusive of those in which the alteration was associated with valvular disease, or adhesion of the pericardium. An analysis of 250 cases of granular degeneration, drawn from the St. George's books, gave 48 per cent. as the proportion of cardiac enlargement.

Dr. Grainger Stewart gives 46 per cent. as the proportion of cardiac hypertrophy with this form of renal disease.

It is probable, however, that all these statements fall far short of the actual proportion of ventricular enlargement in association with granular degeneration. They take no account of the slighter degrees of the change, which from its nature are apt to be overlooked. Since I have had my attention particularly directed to this subject I have scarcely seen an instance in which, if the renal state was distinctly recognized, whether after death or in life, some degree of cardiac hypertrophy was not also apparent. I have, in fact, got to regard simple cardiac hypertrophy as one of the most important diagnostic signs of renal fibrosis.

This increase of bulk in the left ventricle was known to Dr. Bright, and was attributed by him to an alteration in the blood, which caused it to pass with difficulty through the capillary vessels, and thereby called for increased efforts on the part of the muscle. There can be no doubt that this explanation is founded on sound principles. It is not necessary to cite instances of the same sort of action under other conditions. It is well known that the passage of blood through the capillaries is hindered by the presence of matters which it ought not to contain. The retention of blood in the lungs, and its accumulation in the right side of the heart, is a familiar result of impeded respiration, and appears to be due to the presence in the blood of materials which should have escaped with the breath.

The cardiac hypertrophy occurs only exceptionally with the lardaceous change, but it takes place regularly both with the tubal and the interstitial if only they be sufficiently chronic. And if sufficiently chronic the tubal disorder is almost certain to have become associated with more or less secondary fibrosis. The adjustment of the strength of the ventricle to the condition of the blood is necessarily a work of time. The change is, perhaps, generally not more than is called for by the wants of the system, though too often more than can be borne by the thickened but weakened arteries. The cardiac state seldom attracts notice by its symptoms, though at once evident to auscultation.

To this statement, however, there are exceptions ; the enlargement of the heart may be connected with cardiac distress, dyspnœa, pulmonary and other changes such as primary heart disease might give rise to, and finally take a prominent part in the development of renal asthma. The simply hypertrophied heart usually gives sounds which are merely distant, or if otherwise altered are so only in tone or reduplication; the second sound may be accentuated, both it and the first reduplicated, results as they have been explained, of the unnatural and unequal repletion of the several great cavities; but there is usually no murmur unless the valves be diseased; exceptions to this rule, however, occur in which a marked murmur, such as belongs to mitral regurgitation, may be audible while the tension is high, to cease when by appropriate measures it has been reduced. The stretching of the ventricle probably allows the mitral valve to leak, though under ordinary pressure it may be efficient. I have cited a remarkable instance in which not only was a mitral murmur abolished, but the recurrence of agonizing attacks of renal asthma was prevented by appeals to the bowels and skin to the lessening of a condition of abnormal arterial fulness which was apparent.

The special arterial changes with which the cardiac hypertrophy is associated have received sufficient consideration in other parts of this volume; it only remains, therefore, to refer to the coarse and palpable atheroma which is one of the morbid conditions encouraged by this form of renal disease. An analysis of the *post-mòrtem* examinations of 250 cases of the disease showed it to occur, in sufficient extent to call for notice, in a proportion of 52 per cent.

From whatever cause it arises, atheroma is especially a disease of age. Whatever be the state of the kidneys, it is seldom found in youth in a very marked form. But however early the age at which granular degeneration proves fatal, it will generally happen that a careful examination of the arch of the aorta and the valves of the heart will show some specks of this deposit, perhaps not such as to be of practical importance, but enough to declare the tendency. I could mention an instance in which these were found upon the mitral valve and aorta at the age of six, in association with profuse intertubal fibrosis, though as yet granulation was not. Another, where, with the granular kidney, fatal at the age of 14, specks of atheroma were rather widely scattered.[1] And with every advance of age this affection necessarily becomes more constant and more marked.

HEMORRHAGIC ATTACKS.

With deteriorated arteries, a ventricle of more than normal strength, and blood wanting in fibrine and coagulability, hemorrhage is one of the most frequent results of the disease.

Bleeding from the nose often happens, and sometimes proceeds to an alarming extent; it is more frequent with this than with any other form of renal disease, though very common as a consequence of lardaceous change.

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[1] See work on Diabetes, p. 198.

Vomiting of blood poured out without obvious breach of surface is not an infrequent symptom, and I have known hæmoptysis to occur in the same circumstances, though this is one of the rarer accidents of the disease. Purpura is sometimes developed, though this again is infrequent; and a similar statement may be made with regard to menorrhagia.

But the most disastrous way in which the hemorrhagic disposition shows itself is by extravasation within the skull. In the table on page 129 there are three instances in which persons who were under treatment for granular degeneration of the kidneys were attacked with sanguineous apoplexy. But this statement gives a very imperfect idea of the alliance between the two diseases. The table includes only patients who were under observation with recognized albuminuria, and who had generally sought admission because the disease was manifesting itself by symptoms of a chronic kind, tending probably to some other ending. Those who die of apoplexy are apt to be struck, not in the wards of a hospital, but while they are going about making use of what health they have.

If we look at the relationship in another direction, and consider the state of the kidneys in all who have died of apoplexy, we shall find facts which may throw light on the question. During the course of twenty years 75 victims of apoplexy were examined in the dead-room at St. George's; of these 31 were described as having the kidneys in a decided state of granular degeneration. Dr. Ridge Jones[1] has also examined the hospital records from the apoplexy point of view, and I am able to add his testimony on this question. Dr. Jones states that of 36 cases of fatal apoplexy, in which the *post-mortem* examination was made with sufficient completeness to allow of conclusion as to the state of the kidneys, there were 29 in which those organs were extensively diseased. In 24 the organs were described as being small, hard, granular, with their cortical substances diminished—in some instances to the thinness of a shilling. Hence, it appears that it is rather under the fact to state, that of fatal attacks of apoplexy one-half are preceded by granular degeneration of the kidney.

The cerebral is the direct result of the renal lesion. Where the renal state has been induced at an exceptionally early age it is not unknown for the apoplectic issue to be equally hastened, and even to occur in childhood.[2]

We can recognize three circumstances consequent upon or connected with granular degeneration which must assist in causing rupture of the cranial arteries. The vessels themselves are weakened by the special albuminuric change and by atheroma. The force of the left ventricle is increased by hypertrophy. The pressure of the blood upon the arterial walls is further increased by the obstruction in the capillaries, consequent upon the alteration in the blood. The last cause is less evident to the senses than the others, but it is hardly possible to doubt its existence.

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[1] See papers on Apoplexy, by Mr. Thomas Jones, British Medical Journal, 1862.

[2] See work on Diabetes, p. 195.

Another result of the same chain of circumstances is to be found in the derangement of vision which so frequently accompanies granular degeneration. Like epistaxis, apoplexy, and other hemorrhagic attacks, it is associated with the degeneration and rupture of minute arteries.

In the 68 cases of granular degeneration of which the symptoms are given in the table, there are five in which dimness of sight or total blindness followed as a consequence of the disease. A connection between albuminuria and amaurosis has long been recognized. As is the case with apoplexy, the retinal accident is generally associated with the form of renal disease at present under consideration.

The usual concurrence of the granular kidney with the albuminuric retina, as with the hypertrophied heart, formerly led me to think that there was a pathological connection between the intertubal change and the process by which the eye suffered. But though we find few if any exceptions to the rule which connects some measure of renal fibrosis with the retinal change, yet the fibrosis may be secondary and rather an evidence of chronicity than of change of any particular sort. The visual disorder has been known to ensue upon albuminuria primarily lardaceous or primarily inflammatory. It occurs with the greater frequency in connection with the granular kidney, probably not by reason of any special alliance or agreement in kind between the renal and the retinal change, but from the protracted enhancement of vascular tension which this as compared with any other kind of renal disease involves, and the consequently greater regularity with which it becomes associated with the cardio-vascular change of which the retinal is but a part. I have elsewhere given the reasons which have led me to believe that the retinal is not an independent portion of a systemic disease in which the kidneys are also involved, but a direct consequence of their glandular insufficiency.

The retinal disorder is a sign, I believe, always of advanced disease, though it is not infrequently the first sign of the disorder which is recognized.

Inflammatory Attacks.

As always happens with albuminuria, whatever the condition of the kidneys, the sufferer is often called upon to pay his debt before it is due ; to be cut off, as it were, before his time, by some intercurrent disease, to which his disorder renders him prone. His blood is charged with excrementitious materials, which the kidneys have failed to remove, and which act as irritants to certain tissues. There is a morbid tendency to inflammation. As a consequence of granular degeneration, the organs, which are thus disturbed, are not affected with the same frequency as with tubal nephritis. By far the most common disorder of an inflammatory kind, to which these patients are liable, is bronchitis, which happens in about a third of the number; next comes pericarditis. The proportion in the table (p. 129), 16 in 68 cases, refers only to the existence of recent pericarditis. If old false membranes and adhesions had been included the number would have been considerably greater. Pericarditis is the especial characteristic of the granular kidney, and furnishes one of the most frequent means by which the fatal end is accomplished. Slight as is the immediate danger of rheumatic pericarditis, that of albuminuric origin is fatal, while in the recent state, in the majority of instances, it usually occurs in connection with advanced disease and as its last scene. Pericarditis without endocarditis is a characteristic of the granular kidney; nevertheless endocarditis, as indicated after death by recent vegetations upon the valves, occurs with it or separately in a small minority of cases. Of the 68 cases analyzed in the table on p. 129, there was but one in which pericarditis and endocarditis were found together in a recent state. Nor is endocarditis itself of sufficient frequency in connection with this disorder to give any color to its pathology or often lead to clinical results. When valvular disease and the granular kidney occur together the cardiac is commonly the primary, the renal the secondary lesion.

Pneumonia and pleurisy occur next in frequency to the inflammation of the covering of the heart. Erysipelatous and other forms of integumental inflammation happen, but less often than in disorders of which dropsy is a more prominent symptom.

Among the more rare affections consequent upon this disease are peritonitis and a congestive or inflammatory state of some part of the mucous membrane of the bowels. A marked example of this occurrence will be seen in the case of Dodd,[1] in which the intestinal change, but that it was confined to the small bowel, might have passed for the result of advanced dysentery.

Another instance of similar ulceration of the small bowel is given in connection with the detailed report of that already alluded to; and since I made reference to this rare accompaniment of the granular kidney in one of the recent Croonian lectures, my friend Dr. Greenhow has met, as he tells me, with a third. It was that of a young woman who died at the age of twenty-one with granular degeneration, apparently the consequence of scarlatina which she had had three years previously. The ordinary signs of the disorder were well marked, including the cardio-vascular series with retinal hemorrhage and epistaxis. She had obstinate vomiting, and diarrhœa with colicky pain and abdominal tenderness. The last mentioned symptoms were found to depend upon the presence of small ulcers in the lesser bowels, portions of which were partially sloughing, while flakes of membrane like that of diphtheria were found upon the mucous membrane, and in the peritoneum fetid fluid and recent lymph. There was no tubercle in the body, nor beyond the renal disease was there any circumstance by which the ulcerations could be explained.

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[1] See work on Diabetes, p. 197.

It will be seen from these statements that inflammatory affections are much less frequent than with nephritis, and that different organs are selected. Bronchitis, indeed, is common in both. Pneumonia, pleurisy, and peritonitis are characteristic of the more acute disorder; pericarditis of the chronic.

Termination in Cerebral Uræmia.

When all the accidents of the disease have been escaped; when the patient has not been cut off by bronchitis or any other form of inflammation, by apoplexy, or by any other of the dangers which beset his course; when the disease reaches its natural ending, the ending to which every case will arrive if the disease go on long enough, it will terminate by way of "head symptoms."

It will be seen that each form of renal disease affects the nervous system in a manner somewhat peculiar to itself. Whatever the symptoms may be, they depend primarily upon the altered conditions of the blood. Insomuch as the gland is differently altered in each case, it may fail in regard to different constituents of the urine. For instance, the large white kidney fails to remove the water, which with the granular kidney is abundant. Other elements of the urine may also vary, so that with each variety of renal disease we may have the blood poisoned in a somewhat different manner, while the cerebral symptoms which result differ accordingly.

With nephritis the tendency is to convulsive seizures; with the granular kidney the tendency is to a gradual access of tranquil semi-coma. Convulsions sometimes happen, but in the majority of cases the symptoms are of a quiet kind. They generally come on slowly, and are apt to be preceded by vomiting. The patient, who has perhaps been sick on rising in the morning or after his meals, for some time past, begins to find that he is drowsy or restless; that he has headache, giddiness, or a feeling of stupidity. Severe and repeated headaches in these cases must always be regarded as ominous. It may be noticed that his manner has become peculiar, or he may have wandering and delirium, though the latter condition is comparatively rare. Discoloration of the skin also and a certain characteristic odor are among the signs of uræmia and premonitions of the end. I was once leaving the house of a patient who had a gingerbread complexion, an uræmic, almost urinous, smell, associated, as I could not doubt, with advanced granular kidneys. I had ordered, besides other prompt measures, a vapor-bath to be given forthwith. Explaining the perilous nature of the case to the unapprehensive relatives, I referred to the likelihood of fits. In answer to a question "When?" I said it was impossible to say how soon; they might be beginning even while we talked upon the doorstep. And so it literally was. Before the hot-air machine reached the house he was dead.

It is to be noted with uræmic as with puerperal convulsions, that the seizure may be immediately brought on by mental emotion. To mention one instance among several which I could recall, the first attack of a fatal series was immediately sequent upon a somewhat superfluous degree of agitation with which the patient—a gentleman, who for many years had shown signs of granular degeneration—became affected after parting with his wife, long inseparable from him, for the necessary absence of a single day. In such cases the nervous system is charged almost to explosion by the uræmic irritation ; the smallest jar will determine the catastrophe.

After the occurrence of one or more of the signs which have been recounted, or without any warning, the patient may be seized with convulsions or may pass into a quiet stupor, in which he will lie regardless of passing events, but capable of being roused by loud or repeated questions. This state of quiet stupor is very characteristic of renal disease. The pulse is quiet, the skin cool, the temperature below normal, the pupil dilated or natural. There is a peculiar stertor, which increases as the end draws near and the insensibility deepens. This has been described by Dr. Addison.[1] The respiration is generally quick, and is accompanied with labial rather than guttural sounds. Instead of the snoring stertor which follows sanguineous apoplexy the sounds are of a hissing character, to use the expression of Dr. Addison, and are produced by the mouth and lips rather than by the throat and nose. There is no localized paralysis, no inequality between the two sides, but general immobility and torpor. The lateral symmetry of cerebral uræmia is one of its most important diagnostics.

This condition may be preceded or interrupted by convulsions of an epileptic character. Such seizures occur in a large minority of the cases. When present they do not differ from the convulsive attacks which belong to other forms of renal disease.

In some cases coma, or more rarely epileptic convulsions, come on almost suddenly, while the patient is about his usual occupations. It sometimes happens that persons fall down in the streets from this cause. All symptoms of renal disease may hitherto have escaped notice, and the patient may have considered himself well until within a few hours of his death. In such cases the disease may be mistaken for drunkenness, or some other form of narcotic poisoning. The less profound insensibility, the different character of the stertor, and the dilated pupil, must be relied upon as means of distinguishing renal coma from poisoning by opium, while the contents of the stomach and the odor of the breath will give evidence of liquor, should the symptoms be due to drunkenness. In a doubtful case the urine must of course be obtained by means of a catheter, or otherwise.

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[1] Dr. Addison, Guy's Hospital Reports for 1839, p. 3.

It must be observed that patients with granular kidneys are likely to become poisoned by minute doses of opium. In such cases it is extremely difficult to say how far the symptoms are due to uræmic poisoning, and how far to the apparently insignificant dose of the narcotic which the patient has taken.

The condition of the brain after death, under such circumstances, is one which may be briefly described. The noticeable fact about it is extreme anæmia. The large vessels are empty, the gray matter blanched to a pale buff, while the white matter is perfectly colorless, no blood, or scarcely a trace of blood, exuding upon the cut surface. There is often slight excess of watery fluid in the various cavities and interstices; the ventricles and the subarachnoid space contain a little more than usual, but not so as to produce any pressure upon the cerebral substance. The sulci are generally deep, the convolutions prominent, quite unlike their condition when any pressure is exerted from within. The brain is generally firm, as in health.

The anæmic condition of brain which is so evident after death has no doubt a share in the production of the symptoms.

In patients with granular kidneys, as already stated, attacks of sanguineous apoplexy are common. The symptoms need not enter into consideration here, as they are of the well-known and ordinary sort. The occurrence of hemiplegia will be sufficient to distinguish such an attack from an uræmic seizure. In the latter condition hemiplegia is never observed, the limbs are equally useless on each side, and the face and eyes remain perfectly symmetrical. The quiet pulse, pale face, and peculiar stertor of renal coma are further points of difference.

Urine in Granular Degeneration.

The urine is affected in this disease in a manner precisely the converse of what happens with tubal nephritis. It is increased in quantity in the early and middle stages—even to double or treble the normal amount—and if scanty at all is only so after the disorder has lasted a considerable time, or become complicated with tubal inflammation.

The urine is usually bright and clear, and paler than its dilution would account for. When scanty, it is sometimes turbid from urate of soda, but in the majority of cases it remains pellucid. Sometimes it has a peculiar whiteness, not turbid, but not quite transparent.

Early in the disease the urine is free both from albumen and casts. Then a few casts are to be found under the microscope of hyaline or coarsely granular texture. When these have for a time constituted the only direct evidence of the complaint, or possibly when they have not been sought or not found, a trace of albumen appears, which may not be discoverable until the constitutional symptoms are such as to indicate an advanced stage of the disease. So little albumen may be present even to the end that care is needed for its detection. And even the little may not be constant, but discoverable only after sleep or after meals. In most cases, however, it increases with the progress of the disease to a considerable coagulum, though seldom to the amount reached with the more acute disorder.

The specific gravity is generally below the natural standard, varying from 1007 to 1015. It may be, however, where the urine has been diminished in quantity by the occurrence of renal catarrh, as sometimes happens toward the close, that the specific gravity may surmount by several degrees the limit mentioned. As far as my experience has gone the maximum is 1030.

As a rule the acidity of the urine is lessened.

Blood is passed, but with much less frequency than in the more acute disorder. About one patient in ten was found, while in hospital, to pass enough blood to be evident to the naked eye.

When the urine is allowed to stand, casts may generally be found, but in no such abundance as characterizes nephritis.

So long as the disease is uncomplicated by catarrh of the tubes and without hemorrhage the microscopic sediment will consist only of casts. There is no renal epithelium, nor any pus.

The casts which especially belong to this disease, and may perhaps be regarded as peculiar to it, are of coarse granular texture, large, opaque, and conspicuous. These, which are well represented in the plate, are almost invariably present, if casts are present at all.

To hazard a conjecture as to their composition, they may consist of fibrine which has been broken into fragments, or completely disintegrated into a granular *débris* by changes which it necessarily undergoes during its slow passage along the renal ducts. Acetic acid makes these casts translucent, and often shows fragments of fibrine in them. Fibrine, if allowed to decompose in water, breaks down into a coarse granular material closely resembling the casts in question. The granular casts which are found in nephritis, and are composed of altered epithelium, are totally different in appearance.

Besides these casts others are very often present which consist of fibrine in its natural transparent condition; but since such casts occur with every variety of renal disease, they are of less diagnostic importance than the dark granular variety.

Granular degeneration sometimes becomes complicated toward the close of the disease with more or less of tubal catarrh. This intercurrent disorder may be recognized by the nature of the urinary deposit, as well as by the scantiness of the urine and the tendency to dropsy. It may be presumed that this condition is at least partially present when epithelial cells are shed. When this is the case the cells are found as a loose deposit, either in their natural state or with more or less of the character of pus,

PLATE VII., *to face page* 138.

Fig. 1.

Represents all the varieties of Casts which were discovered in the cases of Granular Degeneration upon which the account of the disease is based, making use only of the cases attested by post-mortem examination.

The greater number of casts contain coarse, dark, granular matter; some are of finer texture; a few contain blood-globules or epithelial cells. (See page 138.)

Fig. 2.

Casts from the Lardaceous Kidney. Cases all attested by post-mortem examination. The casts are of two varieties, simple cylinders of fibrine, and casts such as occur with nephritis imbedding epithelial cells. Judging by the casts alone, it might be difficult to discriminate between nephritis and the lardaceous disease. (See also Plate XI.) See page 196.

Plate 7.

Casts—Granular degeneration.

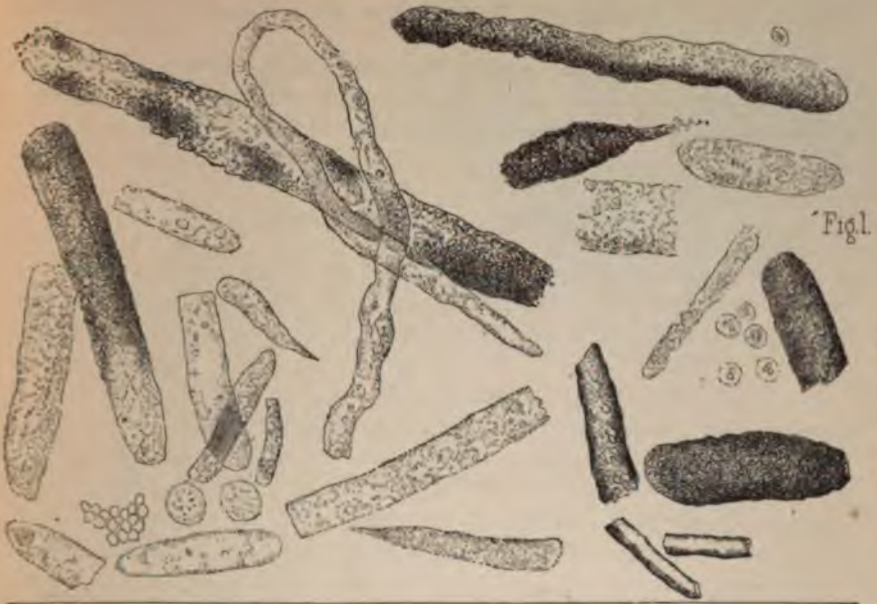

Fig.1.

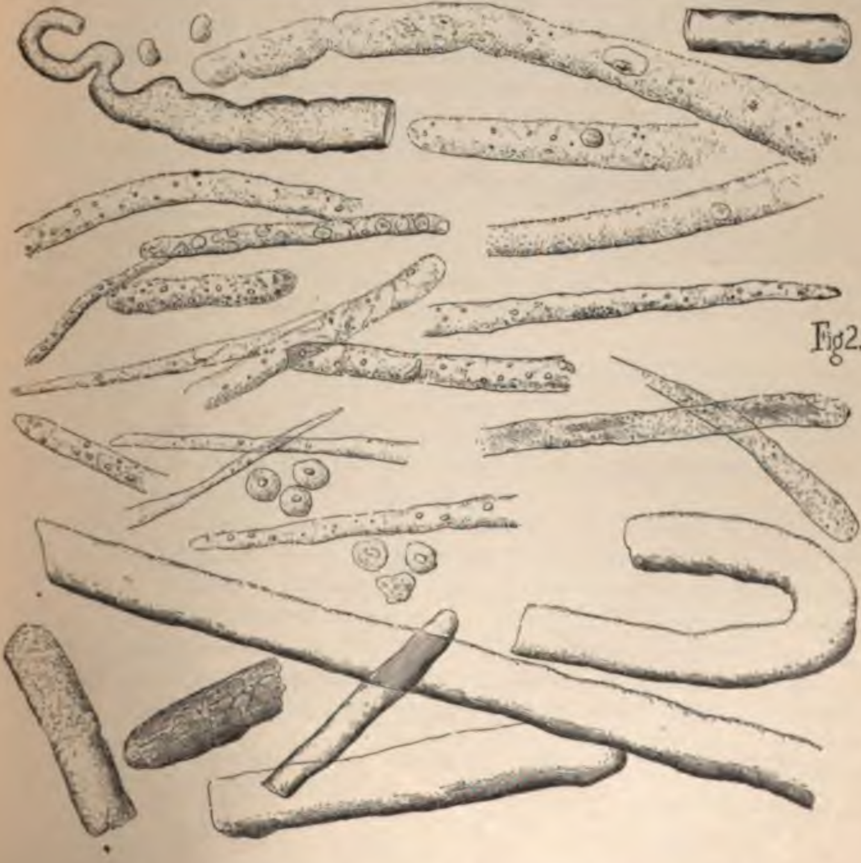

Fig.2.

Casts—Lardaceous or Amyloid Kidney.

besides which some of the casts will imbed cells of the same kind. The casts in such cases are of a mixed sort; some coarse, granular, or simply fibrinous, while others are of the epithelial variety, such as belongs to the tubal disease.

Chemical Changes in the Urine.

Water.—Increased, except in the later stages of the disease, often up to 90 oz. Toward the end the quantity frequently falls below the natural standard. It may even be reduced to 6 or 7 oz. This is characteristic of a very advanced period.

Urea.[1]—Invariably reduced, though not to a great extent, until a very advanced condition of disease is reached. In a case under my own care I traced the diminution of the urea as the disease approached its end, from 23.0 grammes to 8.7 in the 24 hours. The disease may be present for a long time without much diminution of urea, if the urine remains copious. Toward the close, however, the diminution may be extreme. Two cases of the disease are recorded by Rosenstein, both attested by *post-mortem* examination, in which the daily amount of urea fell, before death, in one case to 3.5 grammes, in the other to the exceedingly small amount of 1.0 gramme. This is the extreme of diminution; a more usual reduction is that given by the same author in another fatal case (Wilhelmina Karsten), when the quantity varied from 19 to 12 grammes. From these facts it appears that the variations of urea do not differ much from what takes place with tubal nephritis.

Uric Acid.—In the slighter forms of the disease but little reduced. In the more advanced stages totally absent.

Phosphoric Acid.—Always diminished, much so toward the end. Generally reduced to from half to a quarter of its normal amount. In one case under my own care it fell to .087 gramme before death. This is the lowest amount I have observed in this disorder.

Sulphuric Acid.—This acid is reduced, but to a much less extent than the phosphoric. It is usual to find that, contrary to what is the case in health, the sulphuric acid is more abundant than the phosphoric. In the case alluded to above, near the end of the disease, the sulphuric acid was in five times the quantity of the phosphoric.

Chlorine.—Affected much as the sulphuric acid. Except in the later stages of the diseases, where the urine has become scanty, the chlorine, or chloride of sodium, is little altered. Toward the close the chlorine fell, in the cases already referred to, as low as 1.13 gramme. In a case recorded by Rosenstein the chloride of sodium reached a minimum of 0.7 gramme. In both these cases the urine had fallen much below the usual quantity.

The Alkalies and Earths.—The variations of these constituents is imperfectly known ; but it is ascertained that they are generally reduced, though, since the urine is lessened in acidity, it must be supposed that the acids suffer more diminution. Some details will be found in relation to the case of Dodd (p. 144).

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[1] For the normal amount of the constituents of the urine see work on Diabetes, note, p. 57.

Abnormal Constituents.—The occasional presence of blood has already been discussed. It is not necessary to say more about the albumen than that it is often absent during the earlier stages of the disease, and subsequently may be present only in very small quantity. Sometimes it is copious, but it seldom reaches the amount seen with tubal nephritis. It does not appear that the stage or extent of the disease can be judged of by the amount of this substance. The kidney may be extremely disorganized, while the albumen only amounts to a trace. In some cases, where it has been abundant during the process of the disease, it diminishes toward the close. The amount depends much upon the association of tubal inflammation with the intertubal disease.

The crystalloids, as recognized by the guaiacum test, are sometimes found in great abundance, but are as often totally absent. The more active the disease in the production of dropsy and other symptoms the more regularly is the blue produced by this process.

Stating shortly the chemical changes which the urine undergoes in granular degeneration, they are these. The water is increased, except in the latter stages of the disease ; then it is diminished. The urea, the uric acid, and the phosphoric acid are reduced, but not to a great extent, until the urine has become scanty and the end approaches.

The sulphuric acid and the chlorine are diminished, especially toward the last, but these substances are less influenced by the disease than are the other components of the urine.

Albumen not invariably present, variable in quantity, often in small amount.

Cases.

The following cases are appended as illustrations of the course and symptoms of granular degeneration.

Two of which children are the subjects are placed first, rather as deserving notice from their rarity than as representing the common incidence of the disease.

Unsuspected Granular Degeneration in a Child, Headache, and Vomiting; Death by Cerebral Extravasation at the Age of 12—*Kidneys pervaded with copious Nuclear Growth, in which Capillaries, apparently newly developed, could be Traced—Hypertrophy of Heart and Characteristic Change in Arteries.*

Emily Tillett died at the age of twelve years in the following circumstances. She was brought as an out-patient to the Hospital for Sick Children with pain in the head and sickness, which recurred conjointly about once a week. She was pallid, and her tongue was dry, yellowish, and sometimes brown. Little was ascertained of her antecedents save that she had had several of the complaints of childhood, including scarlatina, which was not known to have been attended by renal sequelæ. An attack of more than ordinarily severe pain in the head, said to have been attended by double vision, led to her admission after three months' attendance.

She walked from her home to the hospital and upstairs to bed. During the following night she was observed to be much excited, screaming and throwing herself about. In the early morning attention was attracted by her increased pallor, and it was found that her lips were blue, the eyes half-shut, the right pupil contracted, while the left was dilated, and the breathing jerking and irregular, while the heart was acting tumultuously. Upon this she rapidly became comatose, and so died half an hour after the alteration was observed.

On *post-mortem* examination a large mass of recently extravasated coagulum was found in an irregularly torn cavity in the posterior lobe of the left hemisphere. The clot, which was about as large as a goose's egg, had broken the surface of the brain, so that part of it was visible immediately on the removal of the dura mater. Inwardly it had similarly ruptured the wall of the left lateral ventricle, though in neither direction had there been any escape. The ventricle contained a small quantity of blood-tinged serum.

The superficial convolutions were generally flattened. The arteries of the brain were free from atheroma, but it was noticed that, especially about the choroid plexuses, they were preternaturally stiff. Microscopic examination showed that the arteries of the pia mater as well as of the kidney were irregular in outline, dotted with oil, and much thickened both in their muscular and fibrous coats. These vessels are figured in the chapter upon the vascular changes connected with renal disease.

The heart weighed 8¼ oz. The left ventricle was contracted and enormously hypertrophied, the valves healthy. The lungs were somewhat congested, but natural in other respects.

The kidneys, which were the only remaining organs otherwise than normal, were very unequal in size, the left weighing 2 oz., the right but half an ounce.

The larger, which was nevertheless morbidly small, had thickened and adherent capsules, which when peeled off (a matter of some difficulty) left a level though not glossy surface, save that at about the centre of the gland was a somewhat reddish cicatricial depression. In section an atrophy common to both cortical and medullary substance was manifest. The usual proportion was maintained, except that opposite the superficial sinking a cone was in absolute contact with the capsule, from a total loss of cortex at that spot. The texture of the cortex was unnatural, of buff and red coarsely intermingled, that of the cones apparently natural.

The smaller kidney nearly resembled the larger in character, though much more shrunk. It had the same thickened and well-nigh inseparable capsule, the same level but scarcely smooth surface, and similar characters, both of cones and cortex, were exposed in section, though so little of either remained that the pelvis was in some places within an eighth of an inch of the capsule, nowhere more than half an inch from it.

Both pelves were large in relation to what remained of the kidneys, but neither of more than their natural capacity. The left ureter was slightly dilated.

With the microscope the changes in the two kidneys were found to be of the same nature. The cortices were occupied and their natural structure in great measure replaced by a copious nuclear formation—a dense congeries of nuclei and fibre—in intimate connection with the arteries and fibrous tissue of the organ, by which the tubes were separated, compressed, and apparently largely extinguished, since wide tracts were seen without any trace of anything but the new growth. This was often seen in insular stellate masses lying at the junctions of several tubes and insinuating its processes between them. It blended itself with and indeed universally pervaded the trabecular matrix of the gland, thickening its strands and narrowing its interspaces. The arteries in relation to the fibroid or nuclear formation were everywhere thickened, and where the growth existed in mass it was seen to be closely intersected by a network of capillary vessels whose walls were of inappreciable thickness, but whose course could be distinctly traced by the blood-corpuscles within them. These vessels were evidently new structures, as parts of the growth in which they lay; their extreme delicacy, together with their close and regular arrangement, made it impossible to suppose that they were primæval remnants, since they could not have survived encroachment and pressure which has been such as to displace or destroy all the more resisting elements of the organ.

Where the tubes remained they were sometimes compressed, elsewhere dilated to twice or thrice their normal width, and irregularly packed with detached partially fatty epithelium.

The two kidneys differed chiefly in the greater abundance in the larger of the swollen tubes, in the smaller the greater extent of the delicately vascular nuclear growth. The appearances here described are represented in the woodcuts on pages 97 and 98.

This case is sufficiently striking in the occurrence, at the early age of twelve, not only of the interstitial disease of the kidney, but of large cerebral extravasation. The hemorrhagic was consequent upon the cardiovascular, and that consequent upon, or in another view associated with, the renal mischief. But the belief that the arterial is merely coincident with the renal change, as part of an universal degeneration, is as difficult to adapt to such a case as it is to imagine the decrepitude of age in the nursery. If we hold the kidneys to have been primarily, and the arterial system consequently affected, the difficulty limits itself to the question why these organs should at this unwonted time of life have become thus diseased. The origin of this renal disorder is often, perhaps generally, obscure. But the urethral dilatation was sufficient at least to suggest that some bygone obstruction to the exit of urine, whether calculous or of some other kind, may have set up the morbid process. The interstitial growth in the kidney was, as is usual, profuse in proportion to the youth of the subject. The fibroid hyperplasia seems, in such cases, to participate in the general activity of nutrition belonging to the period of childhood; we see this not only with young granular kidneys but with the hepatic cirrhosis of early life. But however profuse and active in growth was the fibroid element, the process by which the kidneys were destroyed did not essentially differ from what happens to them commonly at later periods; and the secondary results in general symptoms, arterial changes, and in the fatal hemorrhagic accident were precisely what might have happened at threescore.

Granular Degeneration of the Kidneys in a Boy of Fourteen, sequent upon Vesical Calculus—Symptoms chiefly Cerebral and Gastric, with complete Absence of Dropsy—Hypertrophy of Heart, with characteristic Arterial Changes —Ulceration of Small Bowel, with Peritonitis.

Charles Dodd, fourteen years of age, a boy with a wizened face and a parchment complexion, came into St. George's Hospital with the following antecedents.

At the age of three years he was crushed for stone with apparent success, but never quite recovered his former health or throve as formerly; he remained pale though not especially weak. Three years before admission he became liable to attacks of headache and vomiting, which were especially urgent before breakfast, immediately on getting up. The rejected matter was not bilious, but mostly consisted of white froth. The headache was in the forehead and temples, and was usually relieved by the sickness. The attacks usually came on about once a fortnight. He, however, was able to carry a butcher's tray until in May, 1872, one eye became bloodshot, and the sight of both dim. In the following August a protracted attack of vomiting, followed by a period of unconsciousness with clenching of the teeth and twitching of the right arm, sent him again to the hospital, where for the first time he became my patient, suffering, as was supposed, from a cerebral tumor, a diagnosis due in part to the testimony of the ophthalmoscope.

His complexion, the characteristic "pallor luteus," and the absence of paralysis or lateral irregularity, led at once to the suspicion of albuminuria. There was no dropsy nor had there ever been any, nor had the boy had scarlatina or measles or any serious illness save that produced by the stone and its extraction. The cardiac dulness was increased; there was no murmur. He was thirsty, and passed urine with much frequency and in more than natural quantities. It was pale, of low specific gravity, and wanting in acidity. It gave a scarcely perceptible cloud with heat, but a distinct albuminous line when tested in bulk with nitric acid. Microscopic examination at first failed to show any casts, but repeated with much care on several occasions one strongly defined hyaline cast was at last found with several fragments apparently of the same nature. With these were a few large spherical bodies, some of which were nucleated, others opaquely granular.

The renal origin of the symptoms was thus placed beyond doubt; and the total absence of œdema and manifest hypertrophy of the heart, together with the insidious and apparently causeless access of the complaint, led to the unhesitating belief that the kidneys were of the granular order, notwithstanding the rarity of the affection at the age of fourteen.

A chemical examination of the urine was made a few days later with results which are appended.

Urine for 24 hours ending August 31.

Quantity	2350 c.c.	
Specific gravity	1105.2	
Albumen	2.96	grammes.
Urea	18.8	"
Salts of potash and soda	6.988	"
Salts of lime and magnesia	.615	"
Lime (Ca. 0)	.188	"
Magnesia (2 Mg. 0)	.049	"
Phosphoric acid	1.5	"
Sulphuric acid	.91	"
Chlorine	2.06	"

He was treated at first with a single view to his evident uræmic state; he had small doses of the tartrates of iron and potash with acetate of ammonia as a diaphoretic.

Little change took place in his symptoms until September 1, when he complained of pain in the belly, which was now rather tender and full. He was a little delirious, especially on waking, but had no headache or other nervous symptoms. The bowels were slightly loose, and bleeding at the nose, which he had suffered from previously, had recurred. The yellowish pallor of the face, without tint of pink, and the sharpness of his features were striking, and the latter characteristic became more so day by day, until he assumed a haggard look seldom presented at his time of life. The abdominal pain continued with intermissions and was attributed by the boy himself—he had served a butcher—to "a twisting of the guts." He was generally brisk and sharp by day, though often delirious at night. Fomentations and various local measures of the like purport were used with some temporary but no lasting relief. The bowels were now (September 4) habitually loose, but the motions contained nothing abnormal.

He underwent no further change until September 9, when, while asleep, he had an attack of general but not violent convulsion. In the following night, without any recurrence of the convulsion or any apparent cerebral symptoms, he died somewhat suddenly.

The temperature had been taken frequently in the course of the illness: it varied 99.0 to 99.6.

On *post-mortem* examination the brain was found to be exceedingly anæmic, the fluid under the arachnoid and in the ventricles was somewhat in excess, but the convolutions were not flattened.

The lungs and pleuræ were natural. The heart weighed eight ounces. The left ventricle was contracted and highly hypertrophied, its walls nearly an inch in thickness. The valves were practically healthy, and no sign of endocardial or vascular disease was presented to the unassisted eye save small specks of atheroma upon the mitral valve, on the thoracic and abdominal aorta, the coronary arteries and the vessels of the brain.

The microscope showed the arteries of the pia mater to be greatly thickened both in their muscular and their fibroid coats, and to be in an extreme stage of fatty degeneration.

There was a considerable quantity of yellowish purulent fluid in the peritoneal cavity, and the intestines, especially toward their lower part, were firmly matted together. The lower two feet of the small bowel were deeply ulcerated. The ulcers were various in shape, in some places linear following the valvulæ conniventes around the intestines, elsewhere large, irregular, and confluent. They often exposed the peritoneal coat. The intervening mucous membrane was generally swollen, even so as to present nodular or bossed prominences between adjacent ulcers. It was congested, often to ecchymosis. The connected mesenteric glands were congested.

The kidneys were very unequal in size, though similarly affected as to their structure. The left weighed 3¾ ounces, the right but 7 drachms. The larger was of full and rounded outline, and on the removal of the capsule, which was thickened, a largely granular surface was exposed, the circular prominences being separated by well marked pinkish or gray intervals. In section the cones in some places nearly touched the surface, owing to the superficial shrinking of the cortex, while between the cones no loss, or even some swelling, of this part of the organ was apparent. The smaller kidney was in a similarly granular condition as regarded its surface; it differed from its fellow chiefly in the extreme attenuation of the cortex and a considerable dilatation of the pelvis, which was not only large in comparison to the shrunk remnants of the gland, but absolutely increased as if by distention. The upper part of the ureter was also dilated, but no obstruction there or elsewhere could be found. Examined microscopically, the tissue of the two organs was in practically the same state. The epithelium was not fatty nor markedly changed. Masses of shrunk tubes were seen more or less imbedded in a nuclear fibroid growth which with them formed the intergranular depressions and penetrated the organ wedge-wise from its capsular surface. Other tubes were distended and variously occupied. The arteries were strikingly thickened and imbedded in nuclear fibroid tissue.

This case comprises several points of more than common interest: the youth of the subject of it, the apparent origin of the disease in stone or some process involved in retention or set up by cystitis, the almost sole and even delusive manifestation of the disorder until nearly its close by its cerebral and optic results, and finally the exceptional state of bowel which accompanied its last stage.

The condition of bowel, not dysenteric, for it was confined to the small intestine, without history of typhoid, sign of tubercle, or any circumstances with which we are wont to associate intestinal irritation, is noteworthy if only from its rarity. I have preserved a record of an almost exactly parallel instance which occurred in the person of a young woman who died in the hospital twenty-two years ago. She had the characteristic symptoms of granular degeneration of the kidney, though some of them were hardly then recognized as such. Frequent vomiting, constant nausea, gradual obscuration of sight nearly to blindness, headache, and pale copious and slightly albuminous urine. In addition she had severe colicky pain and diarrhœa. It was found that besides typical granular kidneys and a hypertrophied left ventricle she had as the only other morbid appearance ulceration of the lower half of the ileum with thickening and injection of its walls and attendant peritonitis with the effusion of lymph and turbid fluid. A third case of the same kind, for which I am indebted to Dr. Greenhow, is alluded to in the next chapter. It is hard to explain these cases but on the supposition that the enteric resulted from the renal change. The common circumstances of intestinal ulceration were equally wanting in all. The coincidence, however, of which the instances mentioned furnished the only examples I know, is little more than a pathological curiosity.

Granular Degeneration: Insidious Approach with Alteration of Temperament—Paraplegia—Total Absence of Œdema—Death from an Apoplectic Attack—Examination of Kidneys.

During the year 1867 I frequently visited, in company with Mr. Hatherley, who had charge of the case, a literary gentleman who is the subject of the following account.

This gentleman, who was of spare figure, and of active and abstemious habits, underwent of late years a gradual alteration in temperament which made his friends apprehensive of mental disease. He became depressed in spirits, prone to tears, and unnaturally irritable in temper. He found himself disposed to take alcoholic stimulants in much larger quantities than heretofore. He became weaker and somewhat thinner than usual; and frequently complained of pain in the loins, which was thought to be rheumatic. His intellect remained clear and vigorous, and he continued to pursue his avocations, though with fatigue and difficulty.

One Sunday during September, 1866, while walking home from church, he suddenly became incompletely paralyzed in the lower limbs, so that he staggered in his gait. He reached home with some difficulty, and was seen the next day by Mr. Hatherley and myself. There was then a want of power in both legs, such that he could only walk a few steps, and that with staggering and difficulty. There was decided numbness in the affected limbs, and there was retention of urine. There was a sensation, not amounting to pain, without tenderness, in the lumbar region of the spine.

The complexion was pallid and somewhat sallow. The skin was cool, the tongue clean, the pulse slow (60). There was no trace of œdema. There was no history of gout. It was stated that for some time past the urine had been passed in increased quantity, and with frequency, especially at night.

Some which had been drawn off with a catheter was examined; it was albuminous (albumen $= \frac{1}{12}$), sp. gr. 1014. It was natural in color and acidity. Under the microscope a number of very transparent casts, some faintly granular and some dotted with oil-globules, were seen. There were also crystals of uric acid.

It was sufficiently clear that this gentleman was the subject of granular degeneration of the kidneys. This view was communicated to his family, with a warning as to his precarious tenure of life. The paraplegia was regarded as secondary to the renal disease.

Iodine liniment was now applied to the spine; the bowels were relieved by medicine; and a mixture containing iron and phosphoric acid was given, to which after a time strychnine was added.

The paraplegia gradually diminished, though it never entirely disappeared, and the patient resumed much the same state of health as before the attack. He further improved under the use of weekly vapor baths, and showed so little outward sign of illness that his relatives were inclined to hope that an erroneously grave view had been taken of the case, and quoted, or perhaps I should say misquoted, in support of this hope, the opinion of a medical friend to the effect that "the urine contained no more albumen than it ought."

The urine, however, continued as described; the quantity of albumen and the nature of the casts underwent no change.

As the following summer approached he expressed himself as not feeling so well as in colder weather, and the stomach became irritable, insomuch that he vomited occasionally after meals. The end came suddenly.

One morning in June, when he was thought to be unusually well, and was preparing to go a short distance into the country, he was found on the floor of his bedroom helpless and speechless, but not quite insensible. He had vomited. He was found to have lost the use of the left limbs. He rapidly became quite unconscious, with stertorous breathing, and died about two hours after the beginning of the attack. He had no convulsion. He died at the age of fifty-two.

Permission was obtained to examine the kidneys, but it was not possible to open either the head or the spine. The body was lean ; there was no trace of œdema.

The kidneys were surrounded by adherent cellular tissue and fat. The capsules were closely adherent, so much so as in some places to tear up the surface. The organs were shrunk to about half their proper weight. Their surfaces were covered with closely set granulations, small but very distinct.

On section the cortex appeared to be much wasted. The bases of the cones in some places almost touched the surface of the kidney, while in most parts not more than the thickness of a shilling intervened. The cones themselves were sprinkled with white specks of urate of soda. A few small cysts appeared both in section and on the surface.

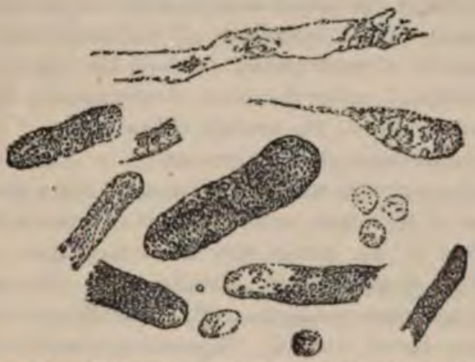
This case is somewhat remarkable, in consequence of the intercurrence of spinal paralysis. I have seen no other case in which this has taken place, nor am I aware that such a complication has been recorded. Although the cord could not be examined, there is little doubt that the lesion was a small extravasation of blood within its substance. The same tendency showed itself in a more fatal form in the final apoplectic seizure. Both occurrences were results of the general arterial degeneration which accompanies the renal disease. That the final seizure was dependent upon extravasation is rendered probable by the presence of hemiplegia, which does not occur from mere uræmic disturbance. In the absence of dropsy the case is characteristic, and scarcely less so in the mental depression. The kidneys were truly gouty ; they contained urate of soda, though no gout had shown itself externally.

In the following case the apoplectic tendency of the disease is also prominently displayed.

Granular Degeneration of the Kidneys—Occasional Œdema—Characteristic Urine—Successive Apoplectic Attacks—Post-mortem Examination.

John Shave, fifty-two years of age, was admitted at St. George's Hospital, November 3, 1858. He was a hotel porter, a temperate person, as he said ; in the habit of drinking "only small beer." He was under the care of Dr. Bence Jones. Two years before he had had an attack of illness with much swelling of the legs, but there had been little œdema since. Three days ago he had been attacked with vomiting, and had since had headache, nausea, and pain in the loins. He denied having had any fit, but his statements, from his uncertain state of mind, could not be depended upon. He was pallid, but there was no œdema. For several days after admission he remained in a sluggish condition, with a peculiar heavy manner, unwilling to speak, but answering rationally when roused. He complained of constant pain across the forehead, and also in the loins; the latter of a very superficial character and affected by movement or pressure. All the limbs could be used, the arms, however, with slowness and apparent difficulty; there was a loss of sensibility in both hands, and to a smaller extent in both forearms.

The fæces were passed into the bed, and the urine upon the floor, apparently by choice. Enough was disposed of in this manner to prove it to be very copious. Some which was obtained was pale, clear, and albuminous (albumen = $\frac{1}{8}$). Acid, sp. gr. 1015. It contained numbers of coarse dark granular casts, of a kind very characteristic of granular degeneration. The tongue was white and tremulous, the pulse 76, full and soft. Cold lotions were applied to the head, a blister was put upon the neck, and a simple saline draught was ordered. Under the influence of these remedies, with frequent purges of compound jalap powder, and latterly a mixture containing nitric acid and iron, he continued to improve, lost his peculiarities of manner and conduct, and almost regained his usual state of health. The urine (November 15) had the same characters, the albumen, however, being in smaller proportion. Casts of the same sort were seen, as well as others of a more transparent kind.



Granular Casts and Cells of Renal Epithelium. From case of Shave.

On December 28 he suddenly fell into a state of complete insensibility. When seen he was unconscious; no paralysis could be made out, but the left arm was colder than the right, and the left pupil was contracted to a mere point. One hand was kept upon the head, as if that were the seat of pain. The evacuations were passed unconsciously. The pulse was weak, 72. After active purgation a slight gleam of consciousness returned, but now (January 1) it was observed that both pupils were contracted, and the right eye squinted inward. There was no paralysis of the limbs. He went on seeming rather to improve, until the evening of the 6th, when he had another fit, which caused his death before the following morning.

At the *post-mortem* examination the kidneys proved to be much shrunk. The capsules were thickened and adherent. The surfaces showed numerous light-colored granules, of rather large size, set upon a purplish ground. On section there was a good deal of fat around the pelves. The cortex was diminished and contained many white spots, as if the same granules which appeared on the surface were scattered through the substance. There were also numbers of small cysts in the cortical part.

One of the kidneys was tested as to its power of conveying water by the vessels, in a manner already described. It was found that only about one-third of the quantity of water passed through the organ, which a healthy kidney should transmit under the same circumstances, the amount being 40 oz. 5 dr., against 119 oz., the average of health.[1]

The large arteries were visibly narrowed.

Under the microscope it was found that there was a general increase of the fibrous tissue of the organ, around the blood-vessels and between the tubes. The tubes in the cortex were filled with dark granular matter, partly composed of closely-packed epithelial cells. The straight tubes, in some cases, had the same contents as the cortical, while others contained transparent fibrine dotted with oil. Separate epithelial cells obtained from the cortical part were perfectly natural.

The vessels of the brain were extremely atheromatous. There was a good deal of blood extravasated in the sac of the arachnoid, and in the sub-arachnoid space, over both hemispheres, at base and vertex. The white substance was firm and pale. The septum was pushed over considerably to the right side by a large clot in the left ventricle, which distended its cavity. The blood here was loosely coagulated and black. The right ventricle contained blood-stained serum, but no pure blood. In the right corpus striatum, at its upper and anterior part, was an old cyst, the walls of which were of a brownish color, and its cavity only just perceptible.

The heart was greatly hypertrophied, principally on the left side; it weighed 22 oz. The valves were healthy, as were the aorta and large vessels. The lungs were excessively congested. All the abdominal viscera were healthy except the kidneys.

This case exemplifies many of the symptoms and tendencies of granular degeneration. The abundant pale albuminous urine, the dark granular casts, the absence of œdema, all pointed to that condition. The head symptoms were shown by the *post-mortem* examination to have been due to a succession of extravasations of blood within the cranium; they were such indeed that they could scarcely have resulted from simple uræmia. The vomiting and pain in the head which occurred shortly before his admission probably marked the occurrence of the extravasation which was found upon the surface of the brain. This was spread equally over both sides, and therefore gave rise to no hemiplegia, but to loss of sensation and impairment of movement on both sides alike. The two apoplectic attacks which occurred during the last ten days of his life, and were accompanied by distortion of the right eye and contraction of the left pupil, were apparently due to a smaller and then a larger outburst of blood into the left ventricle. The old cyst which was found in the corpus striatum probably was of a date earlier than our knowledge of the case extended. The advanced atheroma of the arteries displayed in a marked degree one of the tendencies of the disorder, and with the help of the hypertrophy of the heart caused the death of the patient.

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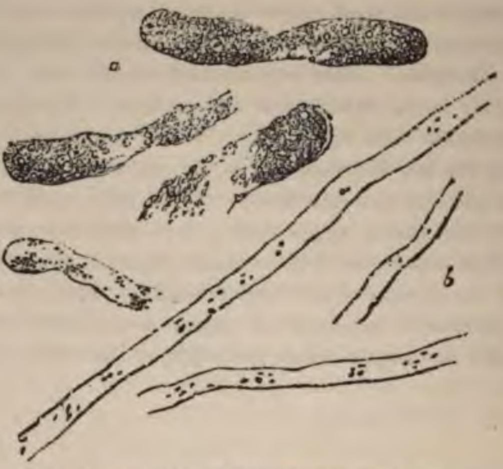
[1] Med.-Chir. Trans., vol. xliii., p. 243.

Granular Degeneration of Kidneys without Œdema or any obvious Symptoms—Bronchitis—Emphysema—Convulsive Seizures—Post-mortem Examination.

Hubert Jutsum, forty-three years of age, a painter, of temperate habits, came into St. George's Hospital, September 21, 1859, under the care of Dr. Fuller.

He described himself as having had bad health for four or five years, though without definite complaint. He never had dropsy, nor pain in the loins, nor observed anything wrong with the urine; for ten years, however, he had nocturnal micturition, passing water about six times in the night. This was the only evidence of renal disease that could be made out from his history. He was thin, and had a worn look. He had been subject for the last six weeks to long fits of suffocating cough, which, with difficulty of breathing, constitutes his chief complaint. He could not lie down. On examining the chest, dry and moist bronchial sounds were heard generally. There was no expectoration. The urine was found to be profuse, pale, and clear; it was acid, sp. gr. 1014. It was albuminous, the coagulum about one-sixth. Casts were found of two sorts, large dark granular and long narrow hyaline. Examined repeatedly, the same casts always recurred. Wine was freely given, with antimonial and stimulating expectorants. A blister was put upon the chest. The breathing gradually became worse, and on October 12 he had a convulsive fit, with a good deal of struggling. He remained afterward in a condition of partial consciousness, sitting up in bed distressingly restless, with cold extremities and a very weak pulse. On the 14th he was quite insensible, lying on his back, with sordes on the teeth and lips. He still swallowed stimulants, which were liberally administered, and to the surprise of everybody he rallied for a few days. On the 28th, however, the difficulty of breathing increased, and with it the orthopnœa. On the 30th he had a second convulsive seizure, after which he remained insensible and gradually sank, dying on the following day.



Granular and Hyaline Casts. From case of Jutsum.

At the *post-mortem* examination the body was found to be much emaciated, quite free from œdema. The heart was large, the left side especially much hypertrophied ; the valves were healthy. The lungs were extremely emphysematous, and there was evidence of bronchitis. The kidneys were contracted, weighing together only 6 oz. The surfaces were exceedingly granular, the capsules adherent, the cortices shrunk. They were typical specimens of granular degeneration.

The liver and spleen were small and hard. The suprarenal bodies, and all the organs which have not been mentioned, were natural.

The brain was not examined.

The gradual failure of health, the pale albuminous urine, the character of the casts, the absence of dropsy, and last, not least significant, the occupation of the man, combined to declare the nature of the renal disease, which was clearly recognized during life. Emphysema of the lungs, to which, in this case, many of the symptoms were due, is not an uncommon complication of granular degeneration. The renal disease in this case had probably been progressing for many years, as appeared from the long continuance of nocturnal micturition, and from the fact that the change in the kidneys was such as could not have been produced in a short period.

Two Cases of Paroxysmal Dyspnœa, with Hypertrophy of the Heart and the General Signs of the Chronic Granular Kidney.

A gentleman farmer, of active habits, and hitherto in vigorous health, became, during the year 1872, what he termed asthmatical; habitually short-winded, and liable to paroxysms of dyspnœa. His urine was found, by Mr. Gorham, of Tunbridge, to contain albumen.

He consulted me about March 20, 1873, having recently had agonizing attacks of dyspnœa in the night. He was often awoke shortly after going to sleep, or was suddenly seized without sleeping at all, with extreme distress, drawing breath with violent effort, absolutely unable to lie down, clutching with violence at furniture, bedewed with sweat, and agonized in expression, and thus remaining for an hour or two in what seemed a mortal struggle. Relief then was obtained, often with much wheezing and frothy expectoration; and then perhaps a few hours' sleep in the sitting posture.

The chest was everywhere sufficiently resonant, excepting in the præcordial region, where was a large extent of dulness, such as indicates a much hypertrophied heart. The sounds were distant, but free from murmur. The impulse was scarcely to be felt. The air entered both lungs very imperfectly, the left most so, the feeble, and sometimes almost absent, breath-sound contrasting remarkably with the ample resonance. The thorax was moved with much effort, the muscles of forced inspiration coming into play; it was lifted rather than expanded; the epigastrium sank with each inspiration. It was thought that the left side expanded less freely than the right.

The urine was rather pale; it coagulated to about one-fifth, and had a sp. gr. of 1012. The legs were slightly œdematous. The face was shrunken and sallow, and his aspect one which contrasted painfully with that of a year before.

The albuminuria, the œdema, the hypertrophy of the heart, with the absence of murmur, at once suggested that the symptoms were, however complicated, primarily renal. Iron, digitalis, and vapor baths were given. With these the œdema disappeared, as likewise did the albumen, and the suspicion that the kidneys were the starting-point of the mischief.

The intermittence of the albuminuria suggesting—fallaciously as it turned out—that the kidneys were but temporarily congested, as a secondary result of venous obstruction, the source of the disorder was sought elsewhere. It was clearly not simple asthma—the dyspnœa, though aggravated paroxysmally, was never absent—and mechanical obstruction to the admission of air, such as would be caused by a growth, appeared to be the only possible solution.

The dyspnœa continued—in the day he usually breathed about 32 in the minute, and with constant heaving effort. The impulse of the heart was found about 1½ inch outside the left nipple; and here and between this spot and the median line was a loud marked reduplication of the fir sound, giving the rhythm of a galloping horse. This reduplication w heard latterly at all times ; earlier only during the nocturnal paroxysms.

He obtained some relief from anti-spasmodics, more particularly opiat but he grew thinner ; for long periods he never lay down, passing most his time propped up in an arm-chair, or bending forward over a tabl with breathing constantly labored, and frequently in seizures such as ha been described. He gradually sank and died on June 6, after a course more than ordinary suffering. For the last fortnight or so the attacks we absent, though the dyspnœa never was. He wasted and became yello and during the last few days the albumen increased so that the urine b came nearly solid when boiled.

On *post-mortem* examination, which was made by Mr. Warrington H ward, the præcordial dulness was found to be wholly due to enlargeme of the heart. It was not practicable to weigh the organ, but it was ap rently of at least twice its normal weight. The enlargement was entire due to the left ventricle, which was enormously increased in absolute su stance and thickness of wall, while the cavity was larger than natural, that some degree of dilatation was mingled with the hypertrophy. T right ventricle was not increased in any respect. The muscular substan of the heart was fatty, the fibres generally dotted with oil, while in patch nothing could be detected but areolar tissue and large oil-globules. Th interior of the heart was practically natural, though the mitral valve wa spotted with atheroma, as also was the aorta. The lungs were not gene ally congested, though a patch of old pneumonic consolidation was foun at the upper part and anterior edge of the right. They were generall tough in texture, as is sometimes noted in connection with old heart-disease and the fibrous septa traversing them were often markedly thickened. A the apex of the right was a single nodule of cretaceous matter. The bron chial tubes were vascular, and contained much frothy fluid. Each pleur contained about half a pint of clear serum. The other cavities and organ were natural, with the exception of the kidneys, which, though too muc decomposed to allow of minute description, had adherent capsules an granular surfaces, and were much shrunk, especially in their cortices, i which were numerous cysts.

As another instance of paroxysmal dyspnœa, with granular kidneys an cardiac hypertrophy, I may mention that of a country gentleman, to who I was called in November last, suffering, as was thought, from disease the heart. I found an attenuated person, of the age of 56, whose face wa covered with the red blotches vulgarly linked with intemperance, describe by the learned as *acne rosacea*.

His complaint was of agonizing dyspnœa, which for the last ten da had come on in nightly paroxysms, which began early in the night, we attended with the strictest orthopnœa, insomuch that since they began he had never slept but in a chair, and which generally resembled those of ordinary asthma, except in the greater distress and apprehension which they involved, and in their association with much palpitation and sense of cardiac distress. After a term of agony and terror which lasted, at worst, for about two hours, relief would come with the expectoration of more or less frothy fluid, sometimes blood tinged or accompanied with separate sputa of bloody mucus, at the same time the lungs becoming pervaded with coarse, moist sounds.

These soon subsided, and he was left free from dyspnœa, and with natural respiratory sounds, but utterly prostrate in body and mind.

Looking further into the case, it was found that the lung-sounds were quite natural, and the heart free from murmur, though the distance of the sounds and greatly increased degree and extent of dulness gave evidence of enormous hypertrophy. With this the pulse was hard and tendon-like, indicating an extreme increase of tension; and the urine pale, abnormally copious, of low specific gravity, and decidedly though not largely albuminous. The tongue was coated and dry, and there was a peculiar odor with the breath, such as belongs to pressing uræmia. The surfaces of the tibiæ pitted slightly on pressure, but there was no other sign of dropsy.

Enquiring into the antecedents of the attack, it was found that this gentleman had for several years suffered much from dyspepsia and morning vomiting, and occasionally from headache; for the last year the urine had been increased in quantity—as was said, to several times its proper amount—while the patient was proportionately thirsty. Contemporaneously with this marked outbreak of renal symptoms he suffered a bereavement which gave him much mental suffering, led him to drink spirits with unaccustomed freedom, and was followed by a nervous condition approaching, as I was informed, delirium tremens, though it was not possible to say how much due to his trouble and how much to the consolation he had sought.

The treatment resorted to consisted, first, in the restriction of diet, which had been somewhat largely of animal food, the prompt use of elaterium, calomel, and vapor baths, and the cautious inhalation of from three to seven drops of nitrite of amyl on the occurrence of the paroxysm. The results were immediate and marked; the general health improved, the tongue became clean and moist, the hardness of the pulse lessened, the paroxysms became less frequent, and on their recurrence were generally overcome "after a mortal struggle," as the patient expressed it, by the nitrite. In the milder attacks he found mitigation from ammonia and sulphuric ether. The attacks were generally associated with extravagant action of the heart, a sense of cardiac distress, and much increase in the rate and force of the pulse. It was noted that in the early part of his illness, while the arterial tension was high, there was sometimes a distinct mitral systolic murmur, which entirely ceased when, under treatment, the hardness of the pulse had lessened.

Under such measures as have been described, succeeded after a time by the cautious administration of iron, the attacks became less frequent, and for the space of about two months were almost entirely absent, while the general health was so far restored that the invalid could walk abroad and find life a pleasure. The seizures afterward recurred, and on their return proved less amenable than formerly to the nitrite of amyl. The inhalation of small quantities of chloroform, however, seldom failed to interrupt them. After a period of nine months, for the earlier part of which the attacks occurred nightly, and afterward, with the interval of freedom which has been mentioned, on an average about once a week, they finally ceased. But their cessation was coincident with much prostration and general failure, together with, whether as cause or result, some degree of pneumonic consolidation of the lower portion of the left lung. Upon this the patient fell into a condition of delirium with rapid emaciation, and at last sank, ten months subsequently to the accession of the asthma, after a course of suffering unusual both in kind and degree.

No *post-mortem* examination was permitted; but as to the state at least of the heart and kidneys none was needed to affirm the hypertrophy of the one and the granular contraction of the other.

These cases—and I can recall several others, though none in which the dyspnœa was productive of more distress—exemplify one of the less familiar concomitants of the granular kidney. Speaking broadly, the symptom is clearly a result of chronic uræmia, though the mode of its production may admit of doubt. The hypertrophy of the heart and the hardness of the pulse habitually present in such cases are evidence of general cardiovascular change; while the exaggerated cardiac action under the attack shows that the circulating system is primarily concerned or early involved in it. Spasm of the pulmonary arteries is suggested by the nature of the attack, while from another point of view it may be regarded as an eliminative effort on the part of the lungs or bronchial tubes. The intermittent habit of the seizures is, however, suggestive of spasm, whether of the bloodvessels or bronchi; while the cardiac type of the symptoms and the excitement of the heart under the paroxysm seem to point rather to the blood-channels of the lung than to the air-channels as the seat of the obstruction.

In the first case, where the dyspnœa, particularly toward the close, intermitted less frequently than in the second, there was found to be emphysema, together with some fibrous thickening of the lung.

The benefit, in the circumstances which have been described, of chronic remedies for the chronic uræmia is sufficiently marked; and scarcely less so the utility during the paroxysm of anti-spasmodics, ammonia and ether by the stomach, and nitrite of amyl or chloroform by inhalation.

CHAPTER X.

TREATMENT OF GRANULAR DEGENERATION.

In the treatment of this disorder it is necessary to have regard to the fact that it is essentially incurable. The kidney has undergone changes from which there is no return. Tubes have been destroyed, and others that remain are interfered with by the presence of fibrous tissue, which is as far beyond the power of medicine to remove as fibrous tissue which belongs to the original structure of the body. We are, therefore, unable to touch the disease in its organic seat. All we can hope to do is to modify its progress and delay its consequences; and in this way much may be done. Life may be prolonged, in some cases almost indefinitely, while the bodily and mental powers are retained with so little loss, that the patient, though a valetudinarian, may be equal to most of the duties and pleasures of life.

There is much less diversity of opinion as to the treatment of the chronic forms of Bright's disease than of the acute.

It is but seldom that a patient comes under treatment until the disease has caused such injury to the gland as is sufficient to disturb the health in some obvious manner. There has been a gradual access of dropsy, there is troublesome dyspepsia or vomiting, the sight has become impaired, some cerebral disturbance has occurred, there is an indefinite failure of health which renders the patient unable to follow his usual avocations, or perhaps some conspicuous change in the urine has arrested his attention.

The irremediable state of the kidneys compels us to direct our attention not to the disease but to its consequences. We may do something to cleanse the system from the contamination of excrementitious matter, and we may meet, with more or less success, the symptoms which arise in the course of the complaint.

There is probably no part of our surroundings which has so great an effect on the promotion or arrest of granular degeneration as climate; and, not to anticipate details which will be found in a separate chapter, I may say that subsequent experience has fully realized the anticipation which I expressed in the earlier edition of this work as to the beneficial action of the South of Europe or Africa in lessening the results of this disease and retarding or arresting its progress.

I have sent persons with whom the disorder, though giving rise to no symptoms which could unfit them for travelling, has been advanced and threatening, to winter in the Riviera, the warmer parts of Italy or Egypt, with the issue of early general amendment, and such certain though slow mitigation in the special symptoms as to show that the advantage to be expected from change of sky is at least as great in renal as in pulmonary disease. Cure is a word to be used with caution, but I have seen little less; the albumen reduced to a trace, and perhaps that inconstant, and the general health brought up almost to its original level.

To pass to expedients accessible to all and to those first which, if the disease be in a quiescent state, may suffice to maintain comparative health, regard must first be had to the patency of channels of exit other than renal. The secretions, particularly that of the skin, should be kept in an active state. The patient should be completely clothed in flannel, and should take regular exercise such as to produce perspiration. The greatest benefit may be derived from the use, at regular intervals, of a hot air or a Turkish bath. Patients sometimes complain, on commencing, that such baths exhaust them, but they generally soon learn to like them for the sake of their obvious benefit.

They may be given twice a week in an urgent case, once a week if there are no pressing symptoms, while in a patient who has been restored to some degree of health by their action, once a fortnight may suffice. Under their influence the general health improves, and the complexion becomes brighter and clearer. The muddy tint of the face may be often seen to be visibly different on the day following the bath. The vapor or hot air bath is of most service in cases where the urine is copious, or at least not scanty; and is more effective as a means of improving the general health than as a cure for dropsy, though now and then dropsy, connected obviously with granular degeneration, will yield to these measures, though it may have defied all others.

Of at least equal importance is the maintenance, by medicine if necessary, of a regular and free action of the bowels, somewhat in excess of what in ordinary circumstances might be consistent with health. Friedrichshall, Carlsbad, or one of the purging waters may be used periodically, or some aperient salt—the sulphate of potash, or of magnesia, or cream of tartar—may be given alone or together with iron, should this be called for; and an occasional more vigorous cathartic, calomel with black-draught or jalap, may be of marked use when indicated by headache, nausea, or especial hardness of pulse. Purgation lessens arterial tension, and often markedly relieves the troubles of the head and stomach, the frequent obscurity of vision, and the other attendant evils of the disease.

Besides the promotion of vicarious secretion it is often needful to give iron as a means of opposing the anæmiating action of the complaint. But it must be given with caution; I have often seen harm to follow from its indiscriminate or too liberal use. It is generally necessary to guard against its constipating action by the association of some aperient with it. The perchloride of iron, which seems of especial service in renal disease, may be mixed with the sulphate of potash, or of magnesia, or the tartrated iron with the acid tartrate of potash. The tincture of the acetate of iron generally suits well, and may be conveniently given with acetate of ammonia or of potash, if either of these drugs be especially called for. The mineral salts, perhaps the iron alum most effectively, may be given, if the urine contain blood or there be any other indication for an astringent.

As to diet it is necessary to pursue a judicious and medium course. Nitrogenous or animal food increases the formation of urea, while non-nitrogenous or vegetable food diminishes it. Urea is probably formed from the excess of animal food, as well as from the waste of tissue. A meal of animal food has an immediate effect in increasing this element of the urine, while vegetable food has been shown not to have the same action.[1]

The food, therefore, should be as non-nitrogenous as is consistent with the proper nutrition of the patient. A purely vegetable diet has been tried, and found not to succeed, and the anæmic tendencies of the complaint forbid the use of a non-nutritious regimen. At the same time it is obvious that the patient should be kept upon the smallest amount of animal food upon which he can thrive.

Alcoholic drinks should be used sparingly. Claret and the lighter wines perhaps provide it in the least objectionable shape. Malt liquors, if, as often happens, the case be complicated with gout, should be avoided. Gin or whiskey, much diluted, are often of value as diuretics. Among drinks the non-inebriant are often of especial use. One of the alkaline mineral waters, such as Apollinaris, for common use, or Vichy, as a somewhat potent antacid, may be used if the gouty tendency prevail, and one of these, perhaps with a similar natural aperient, may in not a few instances be all the medicines in habitual demand.

With such a basis of treatment in quiescent cases complications must be met as they arise.

Dropsy, depending largely as it does upon anæmia, is perhaps more successfully treated with perchloride of iron than by any other means. If the treatment of chronic renal dropsy were limited to the use of one drug, the choice would fall upon this. But remedies of a different class have their use either alone or with some compatible ferruginous salt. With regard to diuretics—most uncertain where they are most needed—it is not possible to lay down rules of any general application as to their selection. Perhaps the most effective is scoparium, used as a decoction, three or four ounces for a dose. Small doses of digitalis, squills, or juniper may be given with one or the other of the saline diuretics, nitre, or acetate or bitartrate of potass. Nitric ether may take a place in any mixture. Sir

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[1] See Parkes on the Urine, p. 50.

Robert Christison, in speaking of remedies of this character, gives preference to a mixture of digitalis, squills, and bitartrate of potass; and my own experience has led me to the habitual use of a similar concatenation. The inhalation of oil of juniper has been commended, but I have not myself seen any advantage from this mode of using the drug. Cantharides is probably always injurious.

Should œdema resist the action of such medicines, purging and diaphoresis must be enforced. Hydragogue purgatives, as jalap or cream of tartar, may be given occasionally or at regular, but not too frequent, intervals. A compound jalap powder may serve twice a week, or a senna draught as often, to which one or two drachms of bitartrate of potass have been added. Elaterium is a resort for emergencies, and something more. Given repeatedly, but not too often, it will sometimes drain the system of dropsy which has resisted all other measures. Vapor baths are better borne in this disease than in any other renal affection, and though too often ineffective, may be used as a means of keeping down œdema. Acupuncture may be necessary in rare cases; it must be used with the precautions already urged.[1] But such measures, which have for their object the removal of dropsical effusion, are only exceptionally needed. Many patients die without having had dropsy in any form, and others have it only for a short time.

A careful regulation of the diet is of course necessary in all cases of dyspepsia, whether associated with albuminuria or with any other condition. In the dyspepsia which belongs to this form of renal disease much may be done by medicine. The remedies which appear most useful are nitric and hydrochloric acids and strychnia. These may be given with calumba or some other mild bitter. Acids appear to be sometimes indicated by the presence of alkaline or ammoniacal secretion in the stomach. When vomiting becomes frequent it is a very intractable symptom. After mineral acids and strychnia, hydrocyanic acid, creosote, brandy and soda-water, and ice, may be used in succession, with more or less relief of a temporary kind. Dr. Johnson advises the use of warm water as an emetic, so as to wash out the abnormal secretion which keeps up the irritability of the stomach.

A state of semi-coma, with or without convulsion, appears to be the natural termination of the disease. Such symptoms are too often an indication that the disease has passed beyond the power of remedies. In some cases, however, the use of vigorous measures will recall the patient to consciousness and give him a new lease of life.

With the granular kidney it appears that there is a smaller tendency to anæmia than attends other forms of renal disease. The subjects of the complaint are mostly of middle age, when the tissues cease to make great demands upon the circulating fluid, and there is established a tendency to plethora rather than anæmia. Whether from this or any other cause, depletion is better borne than with other renal affections. A vigorous purge of elaterium, the action of a strong vapor bath, or even a free blood-letting, will sometimes rescue the patient from impending death. Probably the cases in which the last measure should be employed are few ; extreme hardness of the pulse may suggest it. Generally the best chance will be afforded by acting vigorously upon the bowels. Half a grain of elaterium, a little croton oil, or any aperient which acts powerfully in small bulk, will answer the purpose.

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[1] See p. 51.

It is not seldom that the comatose state to which this disease tends has come on before its time, in consequence of the administration of opium. Intolerance of this drug is one of the peculiarities of the disease ; doses so small as to be looked upon as safe under any circumstances will sometimes have a poisonous effect. I may instance a case where a patient became comatose after taking five grains of Dover's powder, a medicine which has been stated, though with no great probability, not to possess the poisonous properties of the opium it contains.

With regard to the inflammatory and other complications, it is not necessary to follow in detail the method to be pursued for their relief. Such general rules as may be laid down are chiefly of a negative kind. Opium and mercury must be avoided, while measures of a depressing tendency must be used with caution. With regard to pericarditis, one of the most frequent of the complications of the disease, the careless practitioner is often saved any uncertainty as to its treatment by its remaining undiscovered until the *post-mortem* examination. External applications will of course be used where possible. Bronchitis, one of the most frequent complications of granular degeneration, may be treated much in the ordinary manner. Antimony, in small doses, and in conjunction with ammonia, is given with decided advantage in acute cases ; in the more chronic, ammonia with senega and squills.

With regard to the attacks of asthmatic dyspnœa, which are among the most distressing results of the renal disease, they must be immediately met with sulphuric ether and ammonia by the mouth, and, if necessary, by the cautious inhalation of nitrite of amyl or chloroform ; while their recurrence will be retarded by purging, sweating, and such measures as reduce vascular tension and correct uræmia. Laryngeal œdema, sometimes sudden and alarming, may be met, successfully so far as I have seen, with immediate elaterium, the inhalation of steam, and if needful, the execution of a few small punctures about the epiglottis.

Among hemorrhagic complications apoplexy often occurs, as has been mentioned, in persons who are not recognized as having renal disease, and the treatment will be adopted in ignorance of any such complication. Now that bleeding has become unfashionable, and mercury has lost its repute as a *catholicon*, there appears to be nothing in the ordinary treatment of apoplexy which needs to be modified on account of the co-existence of granular degeneration.

Bleeding from the nose sometimes resists the use of ice and perchloride of iron, and has to be stopped by plugging the nostrils. Most of the cases of epistaxis which are obstinate enough to call for this operation are associated either with hepatic or renal disease.

In bringing to a close these practical observations I must divert to a point which the successful issue of a recent case has brought strongly before me—the saving effect of the induction of premature labor[1] where advancing pregnancy is productive of threatening renal symptoms. These, on the emptying of the uterus, if it be not too long delayed—it may be required about the sixth month—will generally subside in so satisfactory a manner that the sacrifice is well repaid. The albumen will quickly retreat from the urine, and the œdema from the face, and though the retinal trouble if present is apt to be more persistent, head symptoms will cease to threaten.

Some of the points of treatment which have been adverted to are exemplified in the following cases:

Attacks of Loss of Speech, and Subsequent Coma, with Granular Degeneration and Gout—Relief from Blood-letting, Purging, and Vapor Baths.

Henry Armstrong, fifty-five years of age, a coachman in a gentleman's household, a man who had lived freely and had gout, was brought into St. George's Hospital, August 23, 1866, under the following circumstances. It appeared that the man, who was of a sluggish nature and overfed appearance, lay down after dinner, suffering from some headache and discomfort. He went to sleep, was roused with difficulty, and then was found to be unable to speak. At 11 P.M. he was brought to the hospital and seen by Dr. Jones, the resident medical officer. He was conscious when admitted, and could walk, but he was unable to speak. In bed he gradually became comatose, with stertor, the pulse at the same time becoming quick and remarkably incompressible. He was at once bled, and as soon as 10 oz. of blood had flowed consciousness and speech returned. 18 oz. of blood were taken. He was purged with calomel and croton oil, and ice was applied to the head. On the following evening he again became unconscious, and the pulse, which had been softer, resumed its former character. He was again bled, this time 10 oz. of blood being taken; again, while the blood was flowing, consciousness and speech returned. He subsequently had a trifling amount of hemiplegia, affecting the right arm. He was purged by means of a turpentine injection, a draught was given at intervals containing sulphate of magnesia and antimony, and he went on without any recurrence of the symptoms. The urine was pale, copious, highly albuminous, and contained dark granular casts. He remained drowsy, but was conscious, though still speaking with difficulty or not at all. He had next an attack of gout, and when he had been in the hospital a fortnight he had a convulsive seizure, with foaming at the mouth. He soon recovered his consciousness, and from this time gradually got better. He took acetate of iron with acetate of potass, and had a vapor bath twice a week, a proceeding which at first he strongly objected to, but the benefit of which was obvious in the increased brightness and clearness of his complexion on each morning following the bath. Under these measures some little œdema which he had had disappeared, and he became able, by October 16, to leave the hospital and resume his duties. He was made an out-patient, and has, since he left the hospital, taken a Turkish bath once a week with great willingness, so assured is he of the good it does him. He now (May, 1867) has the appearance of health, is as active as formerly in his occupation, and has no complaint evident to himself. The urine retains its character; it is pale, plentiful, and albuminous.

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[1] See paper by Dr. Robert Lee "on the induction of premature labor in cases of pregnancy complicated with albuminous urine, dropsy, and amaurosis."—Medico-Chirurgical Transactions for 1863.

Though this man is still alive, there can be no doubt that he has granular kidney. His indolent habits, with a tendency probably to eat and drink more than is good for him, appeared to have given a more than usually congestive character to the cerebral attacks consequent upon the disease. It is probable, indeed, that a slight extravasation took place in the left hemisphere. At the same time it is clear, from the trifling nature of the subsequent paralysis and the rapid recovery of the patient from each attack, that the symptoms were mainly due to functional disturbance. The case furnishes an example of the good effects of blood-letting, as well as of the symptoms which may be taken to indicate such a line of practice.

Granular Degeneration, with Cardio-vascular and Retinal Changes, and Exalted Vascular Tension—Relief to the General Symptoms, and nearly Complete Restoration of Vision by Evacuating Measures.

A soldier, old at 40, who had seen service in India, and contracted intermittent fever in the vicinity of Cawnpore in 1858, and whose spare frame and parchment complexion were characteristic both of his disorder and of his climatic experiences, was referred to me by Mr. Brudenell Carter as having disease of the retina, of renal origin. It appeared that in the October of 1873 he began to suffer from bilious attacks, as he termed them—headache and vomiting, especially before breakfast—together with pain in the loins. These remained the only symptoms until the following February, when the right eye began to fail, and a month later the left. The loss of sight in each eye was preceded by the repeated appearance of a waving flame like that of a candle, after which all outlines became hazy and dim, to the total confusion of the more distant. Mr. Carter, whose detailed description has, I regret to say, been mislaid, found characteristic and advanced albuminuric changes on both retinæ, and transferred the patient to my care on the 28th of March, 1873.

There was no trace of external œdema, nor ever had there been. The chief complaints were of vomiting and headache. The heart gave a largely increased area of dulness, the apex beat was widely diffused, the sounds were distant, the first distinctly reduplicated at the apex. The pulse was extraordinarily hard, the artery feeling in its unvarying fulness like a tendon. He was drowsy, had much dull headache and nausea, and often vomited, especially in the morning. The bowels were confined, the tongue dry; he was thirsty and without appetite.

The urine was pale, abundant, but slightly acid, and deposited crystals of phosphate of lime. It gave an albuminous coagulum which varied from $\frac{1}{4}$th to $\frac{1}{12}$th. No casts were ever found, though repeatedly and carefully sought.

He was put upon a meagre diet—beef-tea, milk, and a farinaceous pudding. An active hydrargic aperient and a vapor bath—the latter to be repeated at regular intervals—were ordered, and, to continue the action of both, a mixture, every six hours, of sulphate of magnesia, bitartrate of potash, and acetate of ammonia. Under such measures, corroborated occasionally by senna and jalap, the headache and nausea subsided and appetite returned. Some strychnia added to the medicine, and a mutton chop to the diet, with watchful care as to the patency of the alvine and cutaneous exits, helped to re-establish the strength of the patient, and by the 19th, he who on the 1st was scarcely able to lift his head from the pillow, was seen groping about the ward with no further discomfort than his want of sight entailed. And this gradually lessened under the continuance of the treatment; and at the same time the pulse became less tense and the reduplication of the first sound of the heart ceased. A little perchloride of iron was added to the laxative mixture on the 21st of April, and by the 17th of the following month the patient was sufficiently restored to return to his home in Carnarvon, and his duties as orderly-room clerk. The sight was such that he could read a newspaper or a clock face on the wall of the ward. The yellowish dark discoloration of the face had strikingly lessened; the skin had become less dry, the urine less albuminous; the headache, sickness, and nausea had entirely ceased, and the patient recovered a healthy appetite. The arteries, though still tense, were less so than formerly. The general improvement was, in short, as great, though not as striking, as that relating to sight; and, taking with him the means of pursuing his treatment, the patient went away full of confidence in his restored efficiency.

A large number of cases of granular degeneration are modelled upon the lines which are indicated in the foregoing narrative. The origin in intermittent fever is indeed not a common experience, nor was it in this instance by any means ascertained, but other particulars—the attenuation, the complexion, the total absence of dropsy, the superabundance of urine, and the absence of renal deposit, are characteristic of pure fibroid contraction, granular degeneration unmixed with tubal disturbance.

The cardiac hypertrophy, the arterial tension, and the retinal hemorrhage, together tell a tale of wide and threatening changes in the vascular system. The use of measures adapted to relieve the blood-vessels and purify their contents, was well exemplified. The blood in such cases is more tolerant of subtraction than addition. Had iron been early resorted to, only aggravation of the disorder could have resulted. Experience teaches that in the circumstances of this patient, while much parsimony must be exercised in matters of food, stimulants, and tonics, remedies ordinarily of an exhausting kind may be dealt liberally, and with no generally exhausting result.

Albuminuria of Long Duration and Insidious Beginning, dependent apparently on Granular Kidneys—Resort to the South—Restoration of General Health and Diminution of the Albumen.

An American gentleman, 44 years of age, who had been much exposed formerly to vicissitudes of weather, latterly to the influence of too frequent "drinks," and had raised himself from a humble position on a western railway to one of wealth and importance, was brought to me by Dr. Sands, of New York.

He was a stout and burly person, with more of British roundness in his build than of Transatlantic attenuation. It appeared that for two years previously he had had slight œdema of the legs, an increase of which in April, 1871, led to the discovery by Dr. Sands, of albumen to one-third in the urine with hyaline casts and a few blood-corpuscles. Under treatment which comprised among other measures hot-air baths, both the swelling and the albumen diminished, and the patient took a voyage to England with further benefit. When I saw him the following September, but a trace of the œdema remained. The cardiac dulness was increased and the first sound prolonged. The urine varied in albumen, so far as I saw it, from $\frac{1}{4}$th to $\frac{1}{12}$th. Collected for 24 hours it gave a measurement of 1840 c.c., a specific gravity of 1015.0, and 30 grammes as the amount of urea. Under the microscope a few casts were found, chiefly hyaline, or sparsely epithelial. The urine was highly acid, not wanting in color. The general health was but little affected, and the chief point on which I was consulted related to choice of climate. I advised the Riviera or Egypt for the winter, to the nearer of which he forthwith resorted. He stayed in the South of Europe until the warm weather of the following spring, when he went for a few weeks to Vichy. He lived very abstemiously, took hot or Turkish baths regularly, and but little medicine. After his return to the United States I learned from Dr. Sands that he had greatly improved in all respects. He had lost weight, but gained in strength. All œdema had disappeared. The albumen had become so inconstant that at times, especially in the morning, but the merest trace was to be discovered. He was sanguine of recovery, and when I last heard of him was about to proceed for another winter to Florida or Nassau.

The unmarked beginning, the slow accession, and the long continuance of the disease, the age of the patient, and the apparent hypertrophy of the heart, are together enough to show almost beyond question that the change was fibrotic and the kidneys granulating. It is probable that these organs were of the large granular type which we in England so often see among the votaries of beer, in which considerable tubal disturbance is associated with the intertubal hypertrophy. The retention of the coloring matter of the urine, the character of the casts, and the bulk of the patient were consistent rather with this type of the disorder, than with that of which contraction is the characteristic of the gland, and as regards the patient attenuation and diuresis.

It is probable that but for the change of habits and atmosphere, which was happily attainable, the dropsy would have become fixed, and the patient have acquired further complications which would have rendered his course possibly a devious but no hesitating decline.

CHAPTER XI.

LARDACEOUS DISEASE.

GENERAL PATHOLOGY.

THE organic change which is thus definitely though somewhat arbitrarily named is common to, or widely scattered throughout, the whole body; it is recognized after death by the action of iodine upon the affected tissues, and during life by symptoms which are almost equally beyond the reach of misinterpretration. The word "waxy" has, especially by the pathologists of the North, been used to designate the same condition as suggestive of the peculiar translucency which it imparts to the solid viscera. More recently it has been described as "amyloid," from a fallacious analogy with starch or cellulose; and later still I ventured to propose the term "depurative," as indicating a connection with suppurative exhaustion—a term which, however justified by the truth which it proclaims, may be abandoned in favor of the older word lardaceous, in deference to the recommendation of a committee of the Pathological Society, whose suggestion in this respect, in the avoidance of the confusion of terms, will, it is to be hoped, be generally followed by those who desire to refer to the change which has been thus variously denominated.

This affection is not in any sense local. It has its origin in changes which pervade the whole frame, and produces tangible alterations wherever arteries penetrate. Many organs are affected at the same time, or in quick succession, and the kidneys are not exempt from the general influence. The primary change is probably—almost certainly—in the composition of the blood; the next a morbid deposit, which first appears in the walls of the small arteries, and afterward in the surrounding tissues. The change may affect the liver, spleen, kidneys, supra-renal capsules, or lymphatic glands; the entire mucous lining of the alimentary canal; the membranes of the brain and cord; morbid growths the result of former disease; or, in short, almost any part of the body to which blood-vessels go, or which, even without vessels, is under the influence of the circulation.

The change, as it affects the solid parts, consists in the infiltration of the tissue by a new material, which can be detected by the action of iodine. The term "amyloid" was rashly given, in consequence of a resemblance which, as far as regards this reaction, was thought to exist between the morbid product and starch. Cellulose, however, rather than starch, resembles the "amyloid" matter in this respect.

The normal tissues take a yellow color with solutions of iodine; the morbid material a deep reddish brown. The morbid material appears to possess a strong affinity for the reagent; it absorbs it eagerly, holds it tenaciously, and assumes its full color, while the healthy parts take only a faint and superficial tinge. This reaction is represented in plates 8, 9, and 11, and is the most ready and certain mode of recognizing the change. A few drops of an iodine solution poured upon a mucous surface or on the section of an organ give under ordinary circumstances a uniform yellow tinge; if the peculiar deposit is present, it becomes conspicuous by the contrast which its deep brown color presents to the unaffected parts.[1]

It has been stated, that with the addition of sulphuric acid a blue tint is obtained, but this appears to be an error of observation, partly arising from the fact that when sulphuric acid acts upon iodide of potassium—a salt generally present in the test solutions used—a precipitation of iodine takes place, which blackens the tissue.

It is not to be forgotten that any crystals of cholesterine imbedded in the organ may yield a blue color on the addition of iodine and sulphuric acid; cholesterine is often abundantly present in lardaceous organs, though no necessary part of the morbid deposit; and it is not impossible that particles of this material may have, in some instances, given a reaction which with the naked eye has been attributed to the proper lardaceous tissue.

With the certain and ready means of detecting the formation which iodine affords we are able to discern its first appearance in the walls of minute arteries, and trace its increase until it forms a formidable addition to the bulk of the viscera. First observed as thickening the walls of the smallest arteries, and producing an appearance of exaggeration of their transverse fibres, the new matter, which then is transparent and homogeneous, penetrates their coats and gradually works its way into the surrounding tissue.

The changes which result are somewhat different in the several organs. In the solid viscera the new material appears to penetrate the walls of the vessels, be taken up in the epithelial cells, as is well seen in the liver, and saturate more or less the whole structure, with preference for the arterial and secreting elements, to remain as a permanent, or at least a very persistent, addition to their bulk.

These organs, the liver especially, often increase greatly in size, and become hard, gray, and semi-transparent, as if they had been intimately and uniformly infiltrated with white wax, an appearance which has given a name to the disease.

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[1] A solution for the purpose is readily made by dissolving iodine in water, with the help of iodide of potassium, until a solution of the color of brown sherry is obtained. Or the same result is reached by diluting the liquor iodi of the Pharmacopœia.

In the liver the part of the secreting substance first affected is the middle zone of each lobule, that in connection with the hepatic artery; the central area or that of the hepatic vein suffers next; and lastly the outer or portal district. Thus in the liver, as elsewhere, the disorder falls first upon the arteries and their surroundings. The kidneys, supra-renal capsules, and lymphatic glands assume under the morbid process the firmness and waxy translucency which are its general results. The spleen sometimes swells to an enormous size; its malpighian bodies are the especial foci of the change, and often assume the size and translucency of grains of boiled sago. The mucous membranes, which are very liable to the change, especially that of the small intestine, are affected in a different manner. The vessels are altered, as are those in the solid organs, but the exudation, instead of being retained in the tissue, appears to pass off as a secretion from the surface, giving rise to vomiting or diarrhœa, when the stomach or bowels are concerned. Mucous membranes thus affected often have an œdematous look, as if they had been soaked in fluid.

Not only does the new deposit modify, as described, existing structures, but tumors mainly consisting of it have been known to occur.[1] Dr. Murchison describes one instance, and I have seen another.[2] The growths were of the size of a chestnut or plum; they were connected in each case with the cerebral membranes; they consisted of a translucent firm material, like cartilage to the naked eye, which was homogeneous, except that it had a tendency to a fibro-nucleated structure. The characteristic reaction was present in each case.

The disease, then, essentially consists of a general outpouring of a certain material which differs from the proper constituents of the body. It remains to be seen what this material is, and in what circumstances it is thrown out.

It is not needful to advert otherwise than briefly to the hypotheses which associate the infiltrating matter with starch and cholesterine; these views have been conclusively refuted by analyses of affected organs, and in the present day would probably find few supporters. The analyses of Drs. Pavy and Odling,[3] and those of Kekulé, conclusively showed the morbid addition to be nitrogenous; and observations which I will recapitulate appear to warrant the conclusion that it is allied rather to fibrine than to any other nitrogenous principle, and that its deposition in a solid state is connected with a deficiency of the alkali with which it is normally associated, and which is its natural solvent in the blood. Thus, to speak roughly, lardaceous matter in this view is dealkalized fibrine; or, to place the conclusion in a shape which can scarcely, as far as one can see, be gainsaid, lardaceous matter is a nitrogenous compound, allied in its properties to fibrine, which differs from the natural components of the body mainly in deficiency of the salts of potash.

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[1] Pathological Transactions, vol. xiii., p. 3.

[2] Ibid. vol. xiv., p. 264.

[3] Guy's Hospital Reports, 1864.

For a detailed account of the reasons which led to this conclusion I may refer to a paper which I contributed to the fifteenth volume of the "Medico-Chirurgical Transactions." [1] I will now be content to give broadly the more important observations bearing upon the subject, comprising those referred to in the paper, together with some of later date, which have been contributed by others as well as by myself.

Elementary analysis shows the morbid deposit to be practically identical in ultimate composition with albumen and fibrine ; and the fact that it is not soluble in water, but persistently solid in the organ until exposed to the action of alkalies, shows it to be more nearly allied to fibrine than to albumen. A peculiarity of the lardaceous material is its solubility in alkali. To refer to the observations which first led me to inquire into the part played by the alkalies in the morbid process, if a piece of lardaceous tissue be exposed to the action of a dilute solution of potash or soda or a stronger one of ammonia, its power of reacting with iodine is at once and finally abolished, and that without any destruction of the normal tissue or even material injury to its microscopic structure. The deposit has, in fact, been dissolved out by the alkali, while as yet the normal tissue is almost untouched. Dr. Marcet made use of this property of the morbid substance to obtain it for analysis, though alkalies do not separate it absolutely from the healthy tissues. If lardaceous tissue be finely divided and washed with water until all the albumen is washed out, and be then treated with a dilute solution of caustic potash, an enormous amount of material is at once extracted, which on the action of an acid will appear as a white flocculent precipitate, soluble in alkalies, but not in their carbonates. The tissue which has yielded this no longer presents the amyloid reaction, and it is an obvious inference that the matter which has caused it has been extracted. But as the same process will extract a similar substance, though far less plentifully, from the tissue of health, the lardaceous matter is not to be strictly isolated by this means. It is probable, however, that the method can be so managed as to obtain the one with but little of the other. In the substance thus obtained Marcet found on an average of three estimations, 13.7 per cent. of nitrogen, the percentage of nitrogen in albumen being 15.7. This accords with older analyses in showing that the substance is at any rate nitrogenous, and in about the same measure as the protein bodies. Whether it be called fibrine or insoluble albumen is, in the present state of our knowledge, a question rather of words than of fact. That it is not ordinary or soluble albumen is sufficiently evident. And its association with fibrine is marked, among other peculiarities, by the circumstance that in lardaceous disease of the kidney casts are sometimes abundantly formed which apparently consist of this material, and which do not differ, save in their reaction with iodine, from the ordinary casts, the fibrinous nature of which it is not possible to doubt.

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[1] On waxy, lardaceous, or amyloid degeneration. Med.-Chir. Trans. for the year 1867, vol. l.

Proceeding to consider the mineral constituents of the morbid deposit, and taking the liver as best suited to the inquiry, I examined the ash obtained from 100 grammes of the fresh tissue in two series of instances, the one representing the condition of health, the other including various stages of lardaceous infiltration. The results are displayed in a tabular form :

Healthy Liver—Seven Specimens—Alkaline and Earthy Salts in 100 *grammes.*

Case.	Salts of the alkalies.	Salts of the earths.	Potash.	Soda.
1. Liver of an adult, apparently natural..	1.181	.053	.187	.255
2. " " " ..	1.11	.173	.23	.189
3. " " " ..	1.041	.033	.205	.252
4. " " " ..	.995	.016	.214	.192
5. " " " ..	.912	.037	.22	.25
6. " " " ..	.904	.05	.191	.123
7. " " " ..	.893	.041	.215	.164

Average percentages :—Soluble ash, or total alkaline salts, 1.00 ; earthy salts (chiefly phosphate of lime), .057 ; potash, .209 ; soda, .196.

Lardaceous Liver—Eight Specimens—Alkaline and Earthy Salts in 100 *grammes.*

Case.	Salts of the alkalies.	Salts of the earths.	Potash.	Soda.
1. Deep bed-sores. Liver in very early stage..........................	1.075	.062	.208	.177
2. Large vomicæ. Liver slightly affected.	.96	.098	.256	.154
3. Sent from Edinburgh. Highly affected. Rather dry when examined.........	.852	.21	.189	.206
4. From Consumption Hospital. Highly affected. Large vomicæ............	.677	.245	—	—
5. Universally infiltrated. Dysentery, syphilis, etc..........................	.633	.154	—	—
6. Decidedly affected. Phthisis, ulceration of bowels.....................	.621	.18	.163	.153
7. Intermixed with fatty change. Disease of pelvis. Discharge...............	.614	.262	.086	.19
8. Decidedly affected. Suppuration from wrist. Phthisis	.481	.209	.12	.053

Average percentages :—Soluble ash, or total alkaline salts, .739 ; earthy salts (chiefly phosphate of lime), .177 ; potash, .169 ; soda, .155.

In health the mineral constituents show no great variation, the potash in particular ranging from .191 to .230 in 100 parts, and giving an average of

.209. Under disease the total alkaline salts and the potash singly displayed a greater variety, in the slighter examples of the change lessened little, in the more extreme instances lessened greatly. Looking at the averages, it is seen that the disease reduced the total alkaline salts from 1.0 to .739 per cent., the potash from .209 to .169 per cent. The earthy salts, it will be observed, were increased.

Passing now from the liver to another organ, I will give some similar observations made more recently with regard to the spleen:

Saline Constituents in 100 *grammes of fresh Spleen.*

HEALTHY.

Cases.	Salts of the earths.	Salts of the alkalies.	Composition of the salts of the alkalies.				
			Potash.	Soda.	Phosphoric acid.	Sulphuric acid.	Chlorine.
1. Healthy spleen..................	.274	1.054	.322	.115	.118	.215	.096
2. "	.121	1.084	.301	.230			
3. "	.142	1.092					

LARDACEOUS.

Cases.	Salts of the earths.	Salts of the alkalies.	Composition of the salts of the alkalies.				
			Potash.	Soda.	Phosphoric acid.	Sulphuric acid.	Chlorine.
1. Lardaceous spleen. Slightly affected. T. Jackson. Cause unknown.	.216	.772	.194	.227			
2. Lardaceous decidedly, but not extremely. Elizabeth Moss. Probably syphilitic..................	.141	.982	.124	.239			
3. Highly lardaceous. Child with enormous vomicæ..................	.258	.658	.218	.115	.07	.049	.105
4. Not extreme. Abscess of ovary. Phthisis..........................	.201	.886	.245	.184	.258	.034	.09
5. Extensively lardaceous. Disease of spine, with discharge............	.354	.699	.203	.190	—	.071	.099

RICKETY.

Case.	Salts of the earths.	Salts of the alkalies.	Composition of the salts of the alkalies.				
			Potash.	Soda.	Phosphoric acid.	Sulphuric acid.	Chlorine.
1. Rickety enlargement of spleen. Ellen Smith..........................	.094	.989	.283	.196	.055	.025	—

As with the liver, the variation under the lardaceous disease is considerable. The potash is constantly diminished, in some instances by more than half; on an average from a percentage of .311 to one of .196. The soda varies much; it has apparently suffered no loss. The earthy salts were generally increased.

These estimations, which in the comparison they afford between the lardaceous condition and health nearly correspond with those of which the liver was the subject, supply an additional fact in the contrast between the lardaceous and the rickety enlargements, of both of which the spleen is a chosen seat. The rickety condition, which is profoundly different from the lardaceous, though the two have a superficial resemblance and have, indeed,[1] until recently been confused together or very imperfectly separated, is distinguished from the lardaceous by the deficiency not of potash but of lime.

Placing these observations in the order of their making, I will now revert to the liver, and refer to some analyses which were performed by Dr. Dupré at the request of the Pathological Society, with a view of testing the statements which I had ventured to make public. Dr. Dupré ascertained the mineral constituents in 200 grammes of liver tissue in three instances of health and in three of strongly marked lardaceous infiltration. The results are here given calculated for 100 grammes:

Saline Constituents of 100 *grammes of Liver Tissue as removed from the Body.* (*Dr. Dupré.*)

FROM THREE HEALTHY LIVERS.

Case.	Potash.	Soda.	Phosphoric acid.	Chlorine.
1.	.3022	.1000	.2312	.1251
2.	.2420	.1190	.3902	.1223
3.	.3021	.0655	.3670	.0695

FROM THREE HIGHLY LARDACEOUS LIVERS.

Case.	Potash.	Soda.	Phosphoric acid.	Chlorine.
1.	.1729	.1498	.1776	.1516
2. Phthisis. Large vomicæ.	.0913	.1745	.3200	.1557
3. Tubercular excavation of kidney	.1315	.2462	.0967	.1916

The relative deficiency of potash is more marked than in most of the instances I have referred to; and it is to be noted, as was evident with regard to the spleen, that the diminution belonged only, as it evidently related most importantly, to this alkali. It is to be mentioned that in the method used by Dr. Dupré the loss of chloride of sodium which is inseparable from incineration was avoided. Giving the averages as applying to 100 parts, the potash in the healthy liver was .283 per cent.; in the lardaceous .131 per cent. The phosphoric acid at the same time is lessened from a percentage of .329 to one of .198; the chloride of sodium is increased.

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[1] See a paper by the Author in the Medico-Chirurgical Transactions for the year 1869, on "The enlargement of the viscera which occurs in rickets."

In regard to the remarkable deficiency of potash in the diseased formation there are some facts of considerable interest, which bear upon the iodine reaction, to which reference has already been made.

The "amyloid" reaction is associated with the condition of acidity. A piece of tissue which has been soaked in a solution of hydrochloric acid will give a deeper tint with iodine than another which has not. Albumen precipitated by an acid will take and retain the color of iodine, as it will not do except when thus acidified. But the most striking counterfeit of the "amyloid" deposit can be made by operating upon fibrine. This normally exists in alkaline combination, but can be transferred to one in which acid predominates by solution in hydrochloric acid diluted to 6 in 10,000 (see Plate 11).

From this solution it can be recovered by evaporation as a gelatinous substance which will take with iodine the tint which is characteristic of the disease, and which contrasts sufficiently with the pale yellow which normal fibrine assumes under the same reagent.

The reaction is so accurately lardaceous as to lend at least color to the idea that dealkalized fibrine is nearly akin to the morbid product.

The re-alkalization of the material will render it again insusceptible of the lardaceous reaction, and restore its original status in this respect. The affinity of iodine for the fibrine as thus dissolved in acid, and the tint which it imparts to it, may be as well shown in the liquid state. If a solution of iodine be added to one of fibrine, the characteristic compound will fall as a precipitate.

The points of resemblance between this substance and that produced by disease have been further investigated by Dr. Marcet in the report to which reference has been made.

To turn to another substance, the reaction of which with the lardaceous material gives similar indications, I may refer to indigo.

Free alkali destroys the color of sulphate of indigo. If a microscopic section of an organ affected as described be treated with this pigment the alkali in the natural structures will destroy the blue color, so that they will in time fade to a light green or yellow; the amyloid matter, however, will retain for an indefinite period the deep blue of the indigo, and will show in bright and striking contrast with the dull tint of the neighboring parts. I may refer to some plates in the paper already mentioned as examples of this effect.

These facts are sufficient to indicate that the morbid matter is distinguished not only by a loss of potash, as has been already shown, but by a loss of alkalinity. Indeed, the extraordinary solubility of the substance in alkalies would seem to prohibit its deposition from normally alkaline blood. The composition and properties of the infiltrating material cannot but suggest that a deficiency of alkaline solvent, and a separation from the blood of something for the solution of which the means no longer suffice, is a rude ideal of the process in which it originates.

The constant presence of potash in the healthy tissues and its apparently essential bearing upon nutrition would prepare us to expect marked results from its deficiency.[1]

Turning to the circumstances which lead to the results which have been described, and regarding the subject now in a clinical light, it would seem that there are several modes in which the necessary impoverishment of blood may be brought about. Some are within our observation, and can be recognized as progenitors of the organic change with a certainty of which there are not many examples in the sable heraldry of disease. Morbid changes in the darkness which involves their origin are like the men they afflict; it is only in exceptional cases that we can trace their descent.

By far the most frequent cause of the disorder is protracted suppuration. The discharge must be copious and long-continued; it is not necessarily connected with tubercle or with any other constitutional taint, or with disease of bone.

The accompanying table, compiled from cases which have come under the notice of the Author, shows that of 66 cases 51 were known to have been associated with a profuse and long-continued drain of pus, while in five suppuration, though not under observation, was concluded, from the *post-mortem* appearances or other circumstances, to have gone on at some antecedent period. Thus about five-sixths of the cases which fell indiscriminately under notice were associated with this process:

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[1] In the preceding account of the lardaceous deposit I have rather endeavored to show what it is than what it is not. In the face of the direct evidence which has been brought forward, it has not been thought necessary to discuss the starch theory at any length. It may be as well to allude to an opinion, originally promulgated by Meckel, that the new material is essentially cholesterine. But this subject is not nitrogenous, as is the infiltrating matter of lardaceous disease; nor does it give the characteristic reaction with iodine, though with this reagent, together with sulphuric acid, it yields a veritable blue. Found continually in morbid growths and exudations, it is evidently rather an occasional and accidental associate of the lardaceous change than essential to it. In the liver, as was shown by Dr. Marcet, cholesterine is present both in health and disease.

Table showing the Affections antecedent to Lardaceous Disease in 66 Cases under Observation of the Author.[1]

Fifty-one Cases in which there had been Protracted Suppuration.

From caries, or necrosis, with exposure of bone	20
" disease of spine, with psoas abscess	1
" profuse suppuration, consequent upon amputation of thigh	1
" " " " compound fracture of leg	1
" phthisis, with vomicæ	11
" dilated bronchi	1
" destruction of kidney by tubercular excavation (one case with psoas abscess of renal origin)	2
" ulcerated cancer	5
" dysentery, with abscess of liver	3
" ulceration of bowels, not dysenteric	3
" deep and long-continued bed-sores	1
" severe syphilitic ulceration of throat or penis	2
	51

Five Cases in which there was Presumptive Evidence that Suppuration had formerly gone on.

From disease of elbow-joint, for which arm had been amputated	1
" extensive disease of spine, with much curvature and loss of bone	1
" phthisis, with extensive cicatrices	2
" history of dysentery in India, also syphilis	1
	5

Four Cases in which there had been Albuminuria Associated with some other Form of Kidney ease. (?)

From tubal nephritis (?) (much dropsy, urine scanty, highly albuminous)	4

Six Cases in which the Cause could not be Traced.

(In one the patient had been long in India, but was not known to have had dys tery.)

Associated with the stated lesions, tubercular deposit was found in 27 cases; can in 5; evidence of syphilis in 6. There were 30 in which there was no sign either tubercle, cancer, or syphilis.

To this statement, which appeared in the first edition of this work will append another, not drawn especially from my own experience, b which represents that of the *post-mortem* department of St. George's H pital, as recorded by a succession of competent pathologists, from the ti when the preceding table was concluded to the present date.

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[1] This table includes all the cases which have come under the notice of the Aut in which a *post-mortem* examination was made; no selection has been made. Ei of the cases were under the age of sixteen, the rest adults. The greater number curred in St. George's Hospital.

Antecedent Circumstances of 83 *Cases of Lardaceous Disease, compiled from ooks of St. George's Hospital, from the Year* 1867 *to the Year* 1875.

NTINUED OR PROFUSE DISCHARGE OF PUS—FIFTY-EIGHT CASES.

Antecedents.	Total number.	Syphilis present in	Tubercle present in	Cancer present in
bone, caries or necrosis	21	3	4	
ith vomicæ	11	—	11	
" +disease of bone	3	—	3	
" +tubercular excavation of kidney	2	—	2	
" +superficial scrofulous sores	2	—	1	
pneumonia, with large vomicæ	1	—	1	
or glandular scrofulous abscesses	2	1	1	
r disease of kidney	1	—	1	
puration, probably of calculous origin	1			
ovary opening into vagina	1			
or sloughing of limbs	2			
chronic bed-sores	2		1	
cess, unconnected with bone	1			
perations, amputation or excision, with e suppuration	2		1	
cancer of uterus and adjacent parts	2	—	—	2
tumor of labium of uncertain nature	1	(?)	—	(?)
+ syphilis	1	1	1	
ngeal abscess, probably syphilitic	1	1		
ulceration of perineum	1	1		
	58	7	27	2

ACCUMULATION OF PUS WITHOUT DISCHARGE—TWO CASES.

Antecedents.	Total number.	Syphilis present in	Tubercle present in	Cancer present in
lection of pus in peritoneum, general losis	1	—	1	
ng ovarian cysts	1			
	2	—	1	

PRESUMPTION OF PAST SUPPURATION—TWO CASES.

Antecedents.	Total number.	Syphilis present in	Tubercle present in	Cancer present in
is, with cicatrices, but no vomicæ	3	—	3	
n of one kidney apparently by suppura- rocess	1	—	1	
	4	—	4	

SYPHILIS, WITHOUT CONSIDERABLE SUPPURATION—ELEVEN CASES.

Antecedents.	Total number.	Syphilis present in	Tubercle present in	Cancer present in
Distinct syphilitic symptoms or history, without long suppuration	8	8	2	
Syphilis, with tubercle or old tubercular cicatrices in lung	2	2	2	
Syphilis suspected but not ascertained	1	1	1	
	11	11	5	

Cause unknown	8	cases.
Tubercle in	37	"
Syphilis in	18	"
Cancer in	2	"
Neither tubercle, syphilis, nor cancer in	37	"
Disease of bone, without distinction of kind, in	24	"

It will be seen that the conclusions of the earlier table are supported by those of the later. Taking the two together, it seems that of the total number of cases, 149, 111 were connected with ascertained, 9 with presumed suppuration; thus presenting the origin of the disease as connected with this process in four-fifths of the whole number. Tubercle as one of the most frequent of all causes of chronic suppuration was present in a large proportion; in 64 instances more than half of those in which the suppurative process had given rise to the disease. It is to be observed that there is no example of tuberculosis without suppuration as giving rise to the organic change in question, and only one in which it was unconnected with a persistent escape. In this exceptional instance the pus had largely accumulated in the abdomen.

Bone-disease—which perhaps stands next to tubercle, with which it is so often associated, in the frequency and persistency with which it causes suppuration—was present in 47 cases, connected with ascertained or necessarily presumed suppuration in all. Cancer had given rise to the organic change in seven instances, comparatively an insignificant proportion, in all with the intervention of discharge. Passing from these to other causes of discharge and deposit, we come to a class in which suppuration *per se*, not connected with tubercle or with any condition to which the term scrofulous could be applied, with cancer or with syphilis, or with disease of bone of any kind, excluding bone-disease of all sorts, since from some scrofula or syphilis might be inseparable, has given rise to the disease.

This class is represented by cases of amputation immediately necessitated by accident, of compound fracture, of dysentery, of gangrene or bed-sores, of ovarian or pelvic abscess consequent upon labor, of renal suppuration consequent upon stone, of dilatation of the bronchi the result of whooping-cough attended with profuse purulent expectoration. They are sufficient to show that neither is any constitutional malady which could be included in the elastic term cachexia necessary to the production of the disease; nor has it any necessary association with bone; but that suppuration in the abstract is not only an efficient but a prolific source. Apart from suppuration neither bone-disease, cancer, nor tubercle appear to be capable of causing it.

Examining the organs with iodine after death where pus has been discharged freely, and for two or three months, it is exceptional not to find evidences of the lardaceous change, though as yet there may have been no symptoms. Given the suppuration, from whatever cause it spring, some degree of the consequent disorder may be generally predicted, though patients differ widely in their power of resisting this exhausting process.

Apart from the formation of pus, perhaps syphilis stands, if not singly at least as solely recognizable as a definite cause of the lardaceous disorder. There is evidence that the constitutional condition is by itself adequate, however it may be aided in many cases by the suppurative effects of its various lesions. In the series of cases comprised in the two tables there was evidence, either historical or pathological, of this affection in 24 instances—about a sixth of the whole; and it is probable that this proportion would be increased were the disorder more open to observation, and less apt to involve social discredit. In the second table, where the morbid associations of each case are separately stated, it is to be seen that syphilis was sufficient as the cause of the lardaceous change, even though not associated with any discharge of pus, or only with one of insignificant amount. Though constitutional syphilis is, as has been insisted on by many pathologists, an effective cause of the lardaceous infiltration, and though severe cases are sometimes associated with it, yet numerically the instances of the disorder due to this cause bear but a small proportion to those in which it has taken origin in simple suppuration or in suppuration complicated only with tubercle or caries.

Perhaps suppuration and syphilis are the only causes of the disorder which in the present state of our knowledge it is possible to isolate. The statements that it may be produced by alcoholism and rheumatism apparently rest, save where rheumatism is a paraphrase for syphilis, on insufficient evidence. It is nearly certain that the disease has no relation to drink save through suppurative lesions, prominently pulmonary tuberculosis, which drink may have helped to engender. It is none the less likely that the apparently simple alteration of nutrition which constitutes the disease may be brought on in ways not as yet obvious; though the proportion of instances to be distinctly traced to the two cases which have been dwelt upon is so preponderating as to show that other influences, if there be any, which cause the disorder are in their results numerically unimportant.

Among my earlier observations I thought I found reason to believe that the disorder was sometimes due to the long-continued discharge of albuminous urine, but later experience has served to show that where the lardaceous condition is associated with one of the other forms of renal disturbance the lardaceous is usually the primary change.

I think it is to be noted that a tropical climate, in the malnutrition which it engenders, at least predisposes to the morbid infiltration.

Having now regard to the ascertained causes of the disease, and taking them in relation to the character of the morbid product, we may form some conception as to the process by which it is developed.

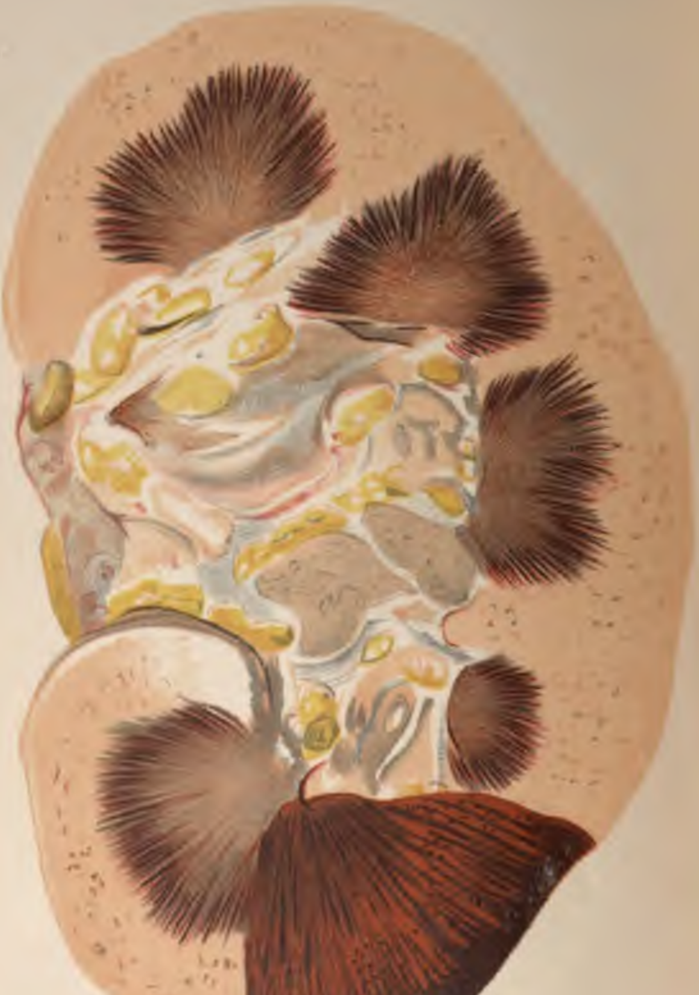
Taking it first in its most frequent and simplest form, as due to suppuration in the abstract, it needs no argument to show that the disorder is a result of exhaustion, not of infection; that it is due, not to the absorption of pus, but to its loss. The deposit, indeed, seems to be so directly produced by the separation of pus from the blood as to suggest that it is nothing more than what remains of the blood after the abstraction of pus, or of the essential part of pus, from it. In this view pus and the lardaceous deposit would be in regard to blood complemental to each other; the blood being divisible into two portions, the one escaping as discharge, the other remaining as deposit.

Pus, however, is not a simple fluid, and it becomes necessary to consider which are its components the loss of which thus affects nutrition. Chemically pus is an albuminous liquid, rich in alkaline matter, abounding, as compared with the blood, especially in the salts of potash. The loss of potash finds its corresponding result in the deficiency of this alkali in the morbid deposit; and it is not improbable that, having regard to the fact that the potash of blood is chiefly contained in its corpuscles, the migration of the white, which constitutes suppuration, is intimately associated with the loss of the essential alkali. That corpuscles are lost under suppuration is as evident as that this process essentially consists in their escape; and it is equally clear that the more are removed the fewer must remain behind.

Looking at the superabundance in the deposit of soda, the alkali of the liquor sanguinis, and the deficiency of potash, the alkali of the corpuscles, a loss of proportion between the blood cells and the blood fluid, and a deposition in a solid form of some part of the fluid excess must suggest itself as belonging to the morbid process. But the mere numerical loss of corpuscles apart from the chemical drain, is not sufficient to explain it.

I have examined somewhat curiously into the proportion of corpuscles to liquor sanguinis under lardaceous disease and its antecedent suppuration; the details are given in Chapter XVI.; they show in the first place, that under suppuration the red corpuscles are somewhat diminished in number, the white, contrary to what might have been expected, increased. With established and progressive lardaceous disease the loss of red is more decided, and these corpuscles are often pale and ill-formed, but the numeri-

Plat



.LARDACEOUS KIDNEY SHEWING REACTION OF IODINE

PLATE VIII., *to face page* 181.

Section of a Kidney affected by the lardaceous change. The organ is much increased in size, the increase affecting chiefly the cortical portion. This has a pale buff color, slightly variegated with spots of vascularity. The peculiar deposit in this case was, as often happens, associated with the accumulation of fatty epithelium in the tubes, hence the great increase of size. A part of the section has been treated with iodine, and shows the effect of that reagent upon the malpighian bodies and the straight vessels of the cones. The former come out as black dots, the latter as dark lines, while the intervening structure is comparatively pale.

The outside of the organ was smooth and buff-colored; it has not been represented.

The kidney was obtained from the body of a soldier who had served in India, and had dysentery and abscess of the liver. He had albuminous urine with dropsy and vomiting, and eventually died in a state of semi-coma. There was extensive hepatization of the lungs.

cal loss is not greater—it is scarcely so great—as occurs with other forms of renal disease, and with the splenic enlargement of childhood, which is not lardaceous, but rickety. The lardaceous result of suppuration is therefore to be attributed, as it would seem, rather to the loss of chemical material than to the escape of organic shapes. But whatever be the effect of the corpuscular loss in bringing about this result of suppuration, the process presents itself as a partial impoverishment of the blood and a deposition of what remains behind in relative superabundance.

But besides the exhaustion of discharge there must be other ways in which the same result may be brought about. A deficiency of income may be similar in its effects to an increase of expenditure, and defective assimilation akin in its effects to a wasting discharge. The attenuated and starved frames of the subjects of syphilis and their obvious inability to transmit nutriment from their stomachs to their tissues, would prepare us to attribute to this disease some special impairment of the blood-making process. It is said that with syphilis the corpuscles are diminished.[1] Whether there be any impairment of the blood-making glands which causes the corpuscles to be insufficiently developed, and the potash they carry insufficiently introduced, may be a matter for further inquiry.

PATHOLOGY OF LARDACEOUS INFILTRATION WITH REFERENCE TO THE KIDNEYS IN PARTICULAR.

From the general pathology of lardaceous disease I will now proceed to the kidneys in particular. These glands are favorite seats of the lardaceous deposit, and, indeed, often display its presence before it can be detected in any other structure. They are affected in a manner which is liable to much complication; not only are they invaded with the peculiar material which, so long as it remains the only change, is attended with increase of bulk but little impairment of function, but the intruded matter appears to act as an irritant, setting up according to circumstances either tubal catarrh, with the attendant symptoms of nephritis or interstitial fibroid thickening, with many of the results, both as regards the organ and the patient, which belong to granular degeneration of independent origin.

Thus, so many shapes belong to the kidneys affected by this disorder that they cannot be more narrowly defined than as responding to the iodine test. They may be swollen or contracted, as tubal accumulation or fibroid contraction have prevailed, and according to the same circumstances, either smooth superficially or variously puckered and granulated.

And, to add another item of variability in the character of organs affected with the lardaceous change, they become fatty in what seems a capricious manner ; the epithelium sometimes loaded with oil to the extreme of possibility, at other times quite free from this addition.

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[1] "The effect of small doses of mercury in modifying the number of the red blood corpuscles in syphilis," by E. L. Keyes, M.D. "American Journal of Med. Science," January, 1876.

These glands are favorite seats for the lardaceous deposit, and, indeed, often display its presence before it can be detected in any other organ.

To describe, first, the changes which are evident to the naked eye they may be thus sketched :

Examined at the earliest date at which the change can be recognized, the kidney is slightly pale and anæmic. If altered in consistence, it is firmer than before. The organ would pass for natural but that the iodine solution dots the malpighian bodies. The surface is smooth, rather pale perhaps, the cortex in due proportion, and every other character that of health. As the disease goes on an increase of size takes place, and the capsule becomes adherent. At this time the cortex is firm, pale, and increased in thickness. It varies in texture. When the increase of size is great there is usually more or less fatty change in the epithelium, and the cortex is of a pale fawn-color, and opaque, like a parsnip. Under these circumstances the kidneys often weigh 10 or 11 oz. each. The largest that have come under my notice weighed, the two together, 23 oz. Where there is no fatty tendency, and the increase of size is less, the cortex often has a pinkish or gray translucency, as if a transparent material were interspersed throughout its structure. This transparent appearance is most observable near the capsule. When the gray effusion is abundant it gives a peculiar firmness and elasticity to the organ. Sometimes the two conditions exist together, buff specks or lines being separated by gray tissue.

The capsule is now adherent, and probably thickened. The surface, still smooth, or marked only by a few curved depressions, has lost the uniform vascularity of the healthy kidney, and has a general bloodless appearance, only variegated by irregular red blotches, or a few stellate vessels. The condition of enlargement belongs to the earlier periods of the disease.

The lardaceous exudation, starting as it does from the blood-vessels, belongs, excepting when it originates within the malpighian body, to the intertubal district, and is early accompanied with changes in the interstitial tissue, such as a low degree of inflammatory irritation might produce, as if the lardaceous matter had something of an irritating property. The early interstitial hypertrophy is succeeded by a contractile process, as with the granular kidney, and soon evidence of contraction appears in superficial depressions. These at first are small and scattered, but gradually increase, and may give rise to extreme changes in the appearance and bulk of the organ. The surface may present various forms of unevenness ; sometimes covered with large smooth elevations, giving an undulating outline ; sometimes irregularly deformed in some places, elsewhere smooth. Sometimes, and this in the most advanced cases, where there is much loss of bulk, the organ has obviously shrunk out of shape ; it has lost more in width and

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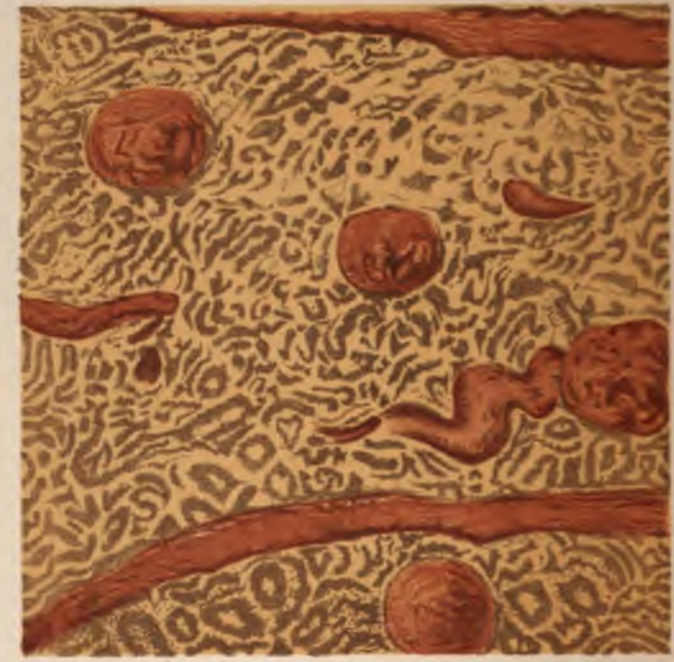

Magn^{d} 35 d.

Section of Waxy Kidney showing action of Iodine

See description of Plates

PLATE IX., *to face page* 183.

Section of a Lardaceous Kidney, showing the action of iodine upon the malpighian bodies and small arteries. These are of bright reddish brown, as seen with transmitted light, while the rest of the section has acquired merely a faint yellow color.

The darker marks upon the section are obstructed tubes. The section was made after freezing with ice and salt.

It was cut from a kidney in a very advanced state of disease, and much contracted, which was obtained from the body of a woman in whom the disorder had followed upon severe syphilitic ulceration of the palate, etc.

She had œdema and ascites, the urine being copious and highly albuminous. There was much diarrhœa and vomiting. Latterly pneumonia occurred.

thickness than in length, and has a lean and attenuated look; and when the capsule is taken off, which is not done without difficulty, the surface has a sandy texture. The regular covering of uniform small granulations, such as follows from granular degeneration, seldom if ever occurs as a consequence of the lardaceous change.

On making a section of a kidney in which this process of shrinking has advanced it is seen that the cortex is diminished, particularly its superficial parts, sometimes the cones being within the thickness of a shilling of the capsule. Whether shrunk or not, it is generally to be seen with the naked eye that the cortex contains numbers of shining dots like specks of glass, which are malpighian bodies enlarged and altered by the disease. Cysts, true to their character of belonging to intertubal disease of the kidney, are common in this disorder. They usually are of very small size.

The great test, however, for the existence of this form of disease is iodine. This holds good in all its stages. Early in the disease the iodine solution simply dots the malpighian bodies, staining them more deeply than the intervening tissues. Sometimes these structures stand out in a conspicuous manner, so as to look like grains of brown sand sprinkled on the section. Usually later in the disease brown lines become visible on the cones, which are straight vessels affected by the disease. Finally, in cases of long standing almost the whole of the substance exposed by section, as well as the surface of the organ, gives the characteristic color.

The deep brown shade—like that of polished walnut wood—of the affected parts affords a striking contrast with the light yellow color of the portions which have escaped (see Plates 8 and 9).

Passing now to the minute and essential changes which the microscope enables us to describe, there are three steps in the disablement of the kidney by this disease:

First, an alteration in the walls of the blood-vessels. Secondly, an effusion through them into the tissue and cavities of the gland. Thirdly, consequent changes both in the interstitial tissue and in the tubes.

In tracing the mechanism of these successive changes the blood-vessels must be first considered. These are affected while yet every other part of the organ is natural, and before there is any symptom of renal disease. The first change is observed in the knot which forms the malpighian body, which becomes somewhat enlarged, and displays the characteristic colors with iodine and indigo. The enlargement is due to a thickening of the vessel, which often gives a nodular outline to the tuft. The size of the tuft increases as the disease progresses, while the capacity of the vessel is diminished. A deposit of fat upon the exterior often helps to produce the sparkling appearance which the malpighian bodies acquire in this disease.

After the malpighian body has become altered as described the same change proceeds to the vessels which enter and leave the capsule. The larger arteries mostly escape, as also do the capillaries and veins. The alteration is often conspicuous in those straight arterial vessels which, after leaving the malpighian capsule, pass through the cones, and have received the name of arteriolæ rectæ. These, as they lie in numbers among the straight tubes, are often distinguished with difficulty from them. They are of about the same diameter, and but for the detection of transverse fibres in their walls would be apt to be regarded as urinary tubules. In many of the affected vessels the transverse fibres of the muscular coat are unnaturally distinct, as if they had undergone a sort of hypertrophy. The walls are thickened and the calibre narrowed. Besides these alterations the affected vessels are readily detected by the peculiar color given by iodine, and by their retaining the deep blue of indigo.

The appearances produced by iodine are shown in Plate 9. For those given by indigo I must refer to a plate I have published in the "Medico-Chirurgical Transactions" for 1867.

These changes in the blood-vessels are commonly succeeded by others in the interstitial tissue, which if the disease be of any standing is apt to become pervaded by superabundant nuclei, to the ultimate formation of fibroid tissue. The hypernucleation is sometimes general, and cannot be distinguished from that which may ensue upon simple nephritis. I had, indeed, prepared a section for representation, but found it so exactly to resemble the drawing at page 23, which relates to a case of simple nephritis, that I forbore to reduplicate the illustration. Often the nuclear and fibroid growth forms most abundantly about the arteries, near the surface, or where the course of one of these vessels has determined the position of a family of malpighian bodies. Around these a broad formation of new tissue is often seen. This, under a low power of the microscope, has a finely granulated appearance (see Plate 10). Under the ⅛th of an inch object-glass the new tissue has a closely nucleated structure, like that presented by new fibrous tissue, or some varieties of fibro-nucleated tumors. This growth may be often traced inward from the capsule, where it is most abundant, along the course of the vessels toward the interior of the gland. It embeds the tubes which lie in its way, and by its contraction reduces them to solid threads, or cuts them up into minute cysts. In a transparent section vestiges of tube-structure may be very clearly seen embedded in the new growth. If these intertubal deposits are of any extent their contractile tendency is generally evinced by an aggregation of the malpighian bodies within them. The distribution of the new growth upon the surface of the kidney is made evident to the naked eye by a loss of level. The situation of each deposit is marked, as has been explained, by a depression, often shallow, smooth, of considerable size, and pinkish in color.

The new fibroid tissue may conceivably arise either from a transformation of the exuded lardaceous matter or as a result of the irritation which it produces; considering, however, that the growth does not react with

PLATE X., *to face page* 184.

Fig. 1.

Section made by Dr. Lockhart Clarke's process, from a swollen lardaceous kidney of which the surface was covered with large curved depressions.

The part represented was opposite a depression. A large expanse of finely nucleated tissue is seen passing inward from the capsule, separating the tubes and surrounding the malpighian bodies. The tubes in the neighborhood contain detached epithelial cells.

At the right-hand side is a part of the new growth, highly magnified, so as to show its fibro-nucleated structure.

Part of a malpighian body appears at one corner; at the other, convoluted tubes. (For comparison, see healthy kidney, Plate V.)

The kidney from which the section was cut was affected in a very characteristic manner. It was obtained from the body of a man who had had an arm amputated for disease of the elbow five years before his death, the immediate cause of which was peritonitis, associated with infiltration of the coats of the duodenum with pus. The liver, like the kidneys, was lardaceous. No renal symptoms were recognized.

Fig. 2.

A section of a characteristic Lardaceous Kidney, made like the preceding.

There is a general increase in the intertubular fibrous tissue. The tubes have lost their epithelial lining, and many contain fibrinous plugs, or casts in their interior. These casts gave the iodine reaction in a most marked manner, taking exactly the same tint as the malpighian bodies. Another section from the same kidney, which displays the effect described, is represented in Plate XI.

The section was cut from the kidney of a man who died of ulceration of the colon.[1] Latterly he had had some œdema, the urine at the same time becoming unnaturally copious. Owing to the disturbed state of the bowels none could be obtained for examination. At the post-mortem the kidneys were greatly enlarged, anæmic, and of a yellowish white color, much like that represented in Plate VIII. They gave the iodine reaction, as did the lining of the small bowel. There was a large suppurating cavity occupying the left side of the belly, and connected with the intestinal ulcers.

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[1] The case is published, as an example of ulceration of the colon, in the "Pathological Transactions for 1867," p. 102, case of C. Roberts.

Plate 10.

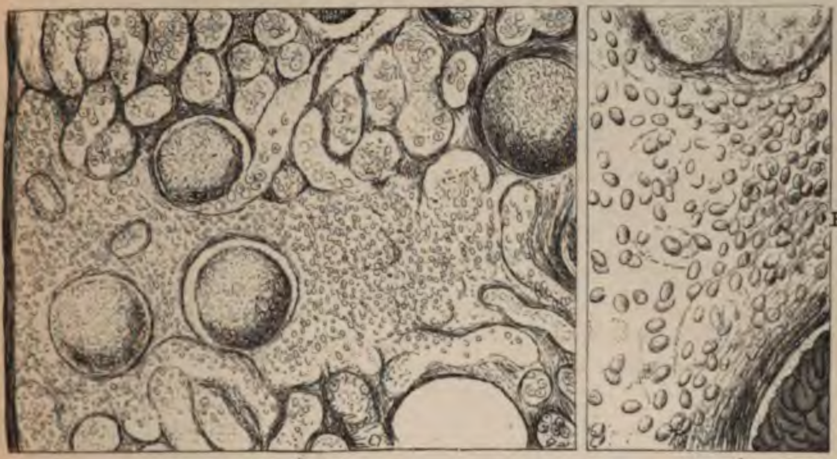


Fig. 1.

Magn^d 75 d. Magn^d 400 d.

Lardaceous kidney, showing intertubular growth.

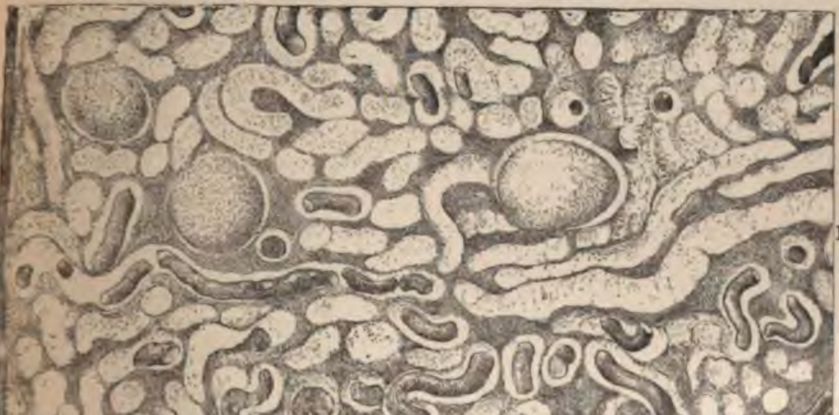


Fig. 2

Magn^d 75 d.

Lardaceous kidney, showing fibrinous casts in tubes.

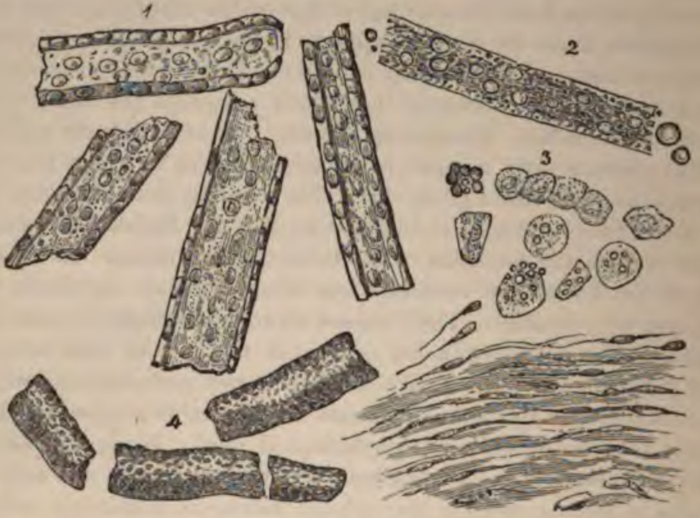
iodine or differ in any respect from the product of simple inflammation, it may with the more probability be referred to the latter cause.

The exudation of lardaceous matter into the substance of the kidney is rather a matter of inference than observation ; all that is to be seen interstitially is vascular alteration and fibrotic change.

But the escape of fibrinous and occasionally distinctly lardaceous matter into the tubes from the exposed vessels of the malpighian tuft is easy of recognition, since the material appears in the tubes and in the urine in the shape of casts. These usually consist of fibrine, which gives no iodine reaction, and exactly resembles that which composes the casts in other forms of renal disease. Sometimes, however, the casts give the reaction in the most characteristic manner, as in the instances to which the accompanying lithogragh refers (Plate 11). The position and abundance of the casts in the tubes is displayed in Plate 10, Fig. 2. Besides this exudation into the cavities, the condition of the tubes requires notice. In the earlier stages of the disease the cortical tubes are, if such an expression be allowed, morbidly healthy. Their normal structure is displayed with abnormal distinctness. The epithelial cells hold to the wall with more than proper constancy, and the cavity continues open and distinct long after death. Kidneys in this state of disease are admirably adapted for demonstration ; they realize quite the physiological ideal. It appears that their peculiar character is due to a fibrinous exudation, which at the early stage transudes in minute quantity through the basement membrane, and glues the epithelial cells to each other and to the tube. The appearance is often such as might result if the epithelial cells had been partially fused upon the wall, which at the same time becomes thickened, probably from some of the exudation becoming incorporated with it. The fixed openness of the tubes is one of the most marked characters of the disease, and one which has its bearing upon the symptoms. Fibrinous casts are abundantly formed and displaced, and yet the epithelial lining of the tube undergoes no disturbance (Plate 10, Fig. 2). Often, however, when the disorder is drawing to an end, supposing that the patient dies of the renal mischief rather than of any antecedent or associated disease, a catarrhal state of the tubes is set up, and they become obstructed, as in cases of tubal nephritis.

I ascertained the condition of the cortical tubes in 32 cases of the disease—some fatal from the kidney symptoms, some from other causes. I found there were 18 in which the tubes were, as described, rigidly open, though often containing fibrinous cylinders. In 14 the tubes were plugged with natural or fatty epithelium.

It is usual in this disease to find, not only that tubes are fixedly open, as has been described, but that they are often distended by fibrinous plugs. They also are often irregularly dilated and constricted, owing to the contractile influence of the tissue around them. When the new growth exists in any bulk the tubes embedded in it are often distinctly cut up into minute cysts. Besides cysts, formed in this way out of tubes, a similar result is sometimes produced by a dilatation of the capsules of the malpighian bodies in the same way as has been described with granular degeneration. Cysts arise, whether the kidneys be granular or waxy, from the constricting action of new growth external to the tubes. With granular degeneration the new formation, though in smaller bulk, has a more powerful contractile tendency, and is therefore a more fertile source of cystic transformation than in the disorder under consideration.



Lardaceous Kidney. 1. Tubes rigidly open. 2. Tubes containing fibrine and oil-globules. 3. Epithelial cells; some fatty, others morbidly adherent to each other. 4 Fibrinous plugs which have come out of some of the tubes. Some new fibroid tissue is also shown.

As an associate of the lardaceous change it is common to find fatty degeneration. The fatty change often precedes the other. After much discharge from vomicæ or elsewhere it may be predicted, without much chance of error, that the liver will be either waxy or fatty. In most cases both conditions will be present. With the waxy kidney oil is often deposited in the epithelium. In 33 kidneys so diseased I found that the epithelium contained oil in 19, in 9 of which the cells were loaded, while 10 were only slightly affected.

Besides the alteration in the epithelium it occasionally happens that, as in granular degeneration, some of the cells become angular and distorted in shape, cramped by the narrowing circumstances of the tube in which they lie.

The peculiar alteration of the smaller arteries, lying next to the change in the blood at the root of the disease, affords a simple explanation of the subsequent changes. These little vessels are so modified as to become penetrable in a morbid degree to the liquid part of the blood which they contain. They retain the corpuscles, but the liquor sanguinis, holding albumen and fibrine in solution, passes through them, both into the interstices of the gland and from the free malpighian vessels into the tubes. As regards the intertubular vessels, the exudation, as it appears to do in every organ when superabundant, stimulates the growth of the interstitial tissue and develops new fibre. A similar escape takes place from the vessels which hang loose in the malpighian bodies. The fibrinous part of their exudation becomes consolidated in the tubes from which it is dislodged in the form of casts. The albuminous portion passes off into the urine without hindrance, and gives its character to the secretion.

With this sketch of the mechanism of the lardaceous change as it affects the kidney it will be easy to see how some of the symptoms arise. The deterioration in the blood-vessels, which is the first tissue-change, though it thickens the walls, makes them leaky, or more than naturally permeable to the fluid they contain. Hence, an unnatural outpouring from the malpighian vessels and an increase of urine, which at the same time is mixed with serum. The tubes remain freely open, and for a long time nothing hinders the passage of serous or aqueous fluid from the malpighian bodies. The fibrinous part of the exudation which passes from them forms the casts which are so abundant. Partly by the plugging thus occasioned, and partly in consequence of a certain amount of tubal inflammation which is apt to come on toward the end of the disease, the tubes become obstructed, and the hitherto copious urine becomes scanty, exemplifying a rule which holds good with every form of renal disease, that the diminution of the urine is in direct proportion to the obstruction in the tubes.

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CHAPTER XII.

LARDACEOUS DISEASE OF THE KIDNEY. CLINICAL HISTORY. SYMPTOMS AND RESULTS.

The disease affects both sexes and all ages.

It is rather more common in males than females, possibly because the former are more exposed to many of the incidents which give rise to it, such as accidental injuries and the effects of tropical climate. Of 61 cases 36 occurred in males, 25 in females.

No age, except the earliest, is exempt from its attacks. Under my own observation it has proved fatal at the age of 5 years, and at every period of of life afterward, up to 70. It is most frequent between 20 and 30, corresponding in this respect with phthisis, with which so many of the cases are associated.

The following case, which is not included in the foregoing statement, as the kidneys were unaffected, shows the earliest age at which, within my own experience, the disease has been detected in any organ.

My colleague, Dr. Gee, called my attention to a spleen which was enlarged, full of translucent bodies like grains of boiled sago, and which with iodine gave the "amyloid" reaction in the most characteristic manner. It had been obtained from the body of a boy two and a half years of age, who had been under treatment in the Children's Hospital for the previous two months with a large abscess in the thigh, which had discharged most profusely. The cause of death was pyæmia. The abscess subsequently proved to have been connected with disease of the hip-joint. No iodine reaction appeared except upon the spleen. Although the disease rarely comes under notice very early in life, there appears no reason why it should not occur at any age where suppuration can have continued.

The following table represents the distribution of 61 cases among the several decades of life. It will be seen that this disease includes almost the whole period of human life, while nephritis and granular degeneration each pertain to one extremity.

The greatest mortality of the disease is later than that of nephritis, earlier than that of granular degeneration:

Table showing Age at Death in 61 *Cases of Lardaceous Renal Disease under the Observation of the Author, Attested by* Post-mortem *Examination.*

Age.	No. of Cases.
From 0 to 10	3
" 11 " 20	11
" 21 " 30	21
" 31 " 40	10
" 41 " 50	10
" 51 " 60	3
" 61 " 70	3
Over 70	0

Total, 36 males, 25 females.

The disorder is easy to recognize during life, perhaps more so than either of the other forms of renal disease. It may be often detected at first sight by the worn and cachectic look of the patient. There are several circumstances which, when they exist together, place the nature of the complaint beyond doubt. The disease follows upon a purulent discharge, or occurs in a syphilitic subject. The urine is albuminous; it often exceeds the natural amount, notwithstanding that œdema may be present. Frequently there is diarrhœa. The liver and spleen are apt to be enlarged. Such are the more salient points by which the complaint may be recognized. But it is not necessary that each of these symptoms should be present. The nature of the disease may be clear, although the urine be scanty, the action of the bowels natural, or the liver remain out of the reach of the fingers.

It may happen that the subject of the renal change is under surgical treatment for disease of bone or joint, and the opinion of the physician is sought, because something in the progress of the case has raised a doubt as to the capability of the patient to recover from a contemplated operation. In such a case the obvious nature of the antecedents renders the diagnosis almost a matter of course. The same may be said when œdema and albuminuria come on in the course of phthisis.

Frequently it happens that the patient is brought under notice solely on account of dropsy, and it is only on careful inquiry that the nature of the preceding disorder becomes evident. It is often found that there is an interval between the cessation of the purulent discharge, with recovery from the original local disease, and the appearance of renal symptoms. Sometimes scars upon the person, or distortions of the spine, will point to the source of the complaint. We may be guided to it by the peculiarity of voice which results from loss of the soft palate, the effect of syphilitic ulceration at some former date, or by some other sign of constitutional syphilis. Sometimes a limb, conspicuous by its absence, will tell a tale of protracted suppuration. In one case which came under my knowledge the disease was apparently due to the discharge which attended the healing of a compound fracture. Or, perhaps, the source of the disorder has left no external mark. It may have been an attack of dysentery, almost forgotten during the various ills of a tropical life; or ulceration of the bowels, tubercular or of some other sort, which no longer exists.

When by such causes as have been assigned, or by others which are less obvious, the tendency to the morbid deposition is established, the various organs are affected in an order which is not always constant.

The first change is generally to be detected either in the kidneys or the liver; in the kidneys more often than in the liver. Whichever of these structures is first affected, it is usual for the other to follow at no distant time. The spleen, the small intestine, the stomach, and the large intestine participate in the disease in something like the order in which they are mentioned. The symptoms which arise are due in chief to the kidneys and the mucous membranes.

It appears that the deposit which is the essence of the disease may take place to a considerable extent before any symptoms are produced. This is continually seen in *post-mortem* examinations of patients who have died of surgical diseases. A small amount of recent deposition, lying as it does within the wall of the malpighian artery, does no harm. It is not until the wall of the vessel has been so damaged as to allow the liquor sanguinis to come through it unaltered that the urine becomes albuminous and the system suffers. The same may be said of the vessels in the mucous membranes. Morbid exudation into the stomach and bowels, as evinced by diarrhœa and vomiting, do not occur until long after the change in the vessels has been such that were the patient to die the iodine reaction would appear. It must be borne in mind that, in the kidney especially, the contractile tendency is a source of injury to the gland, which increases with time. It is owing, probably, to this that the symptoms of renal disease are apt to become apparent after the discharge has ceased.

In order that the deposition should be produced in the vessels it does not appear that a long time is necessary. In one case the reaction was evident upon the malpighian bodies of the kidney when a discharge had only existed for twenty-one days. The patient, a vigorous young man, had had his thigh amputated in consequence of an accident. Most profuse suppuration took place from the stump, and he died on the twenty-first day after the operation. The kidneys were the only organs affected. A much longer time than three weeks is required before such changes can be produced as to make themselves manifest during life. It often happens, as already stated, that the disease does not attract attention until a long time, even years, after the local drain has ceased, or the conditions, whatever they may be, which have given rise to the disease have been reckoned among the past.

When the disease has caused a certain amount of alteration in the kidney the health becomes affected. It is upon the progress of the disease in this organ and in the mucous membranes that the symptoms mainly depend.

The enlargement of the liver and the spleen may be palpable, and yet the health may remain without conspicuous change. As soon, however, as the alteration has progressed far enough in the kidney to render the urine decidedly albuminous the health becomes precarious. The secondary fibrotic changes which occur in this organ render it impossible that the gland affected by them should ever be restored to its pristine condition. If, however, the primary source of mischief ceases before the organ has been intertubally or extensively damaged, it is not easy to say how far it may resume its normal state.

The disease, always chronic, is more especially so when it is found as the consequence of a discharge which has ceased or of a subsiding syphilitic infection. The course of the disorder must usually be counted by years, if we reckon its commencement from the complaint which gave it birth. It is not unusual, however, for it to remain latent until within a few weeks or months of death. If we reckon its duration from the appearance of œdema, or from the symptom which first draws attention to the kidney, we shall find that, though it sometimes passes through many fluctuations of better and worse, and occasionally drags on through many intervals, during which active symptoms are absent, yet that in more than half the cases its course will be counted by months rather than years. This disease is probably more variable in its rate of progress than either of the other forms of renal disease; sometimes, as far as symptoms are concerned, as rapid as tubal nephritis; sometimes in its slowness approaching the character of granular degeneration.

Usually the first symptom of the disease is an increase in the quantity of the urine, with a consequent necessity for nocturnal micturition. This is accompanied by thirst. Œdema then makes its appearance, not with a sudden outbreak, as is the case with nephritis, but with a gradual access. The ankles are observed to be swollen in the evening, even though the urine continues to exceed its normal amount. The swelling, seldom excessive, is apt to invade the peritoneum. The pleuræ and pericardium generally escape; if they become charged with fluid, it is usually not dropsical, but the result of inflammation. At the same time the aspect of the patient is cachectic. He often has the look of one who has been broken down by chronic disease.

There is a marked tendency to inflammation, showing itself most often in the lungs as pneumonia, next in order of frequency as pleurisy; inflammation of the pericardium and peritoneum sometimes occurs, but less often. Pneumonia in particular is more frequent in grown persons from this disease than in either of the other forms of renal malady. Pericarditis is less common than with granular degeneration, more common than with nephritis. Bronchitis can hardly be regarded as a concomitant of this disorder. There is, as with other forms of renal disease, a tendency to erysipelas or to inflammation of the cellular tissue.

Other morbid tendencies mark the course of the disease. Some of these result not directly from the state of the kidney, but from the concurrent progress of the disease in other organs. The diseased vessels of the alimentary canal secrete extravagantly, and give rise to diarrhœa and vomiting. The frequency and severity of these affections are characteristic. Diarrhœa in particular, which seldom occurs with the other forms of albuminuria, is a most common and most fatal result of this. The motions are watery and very frequent; there is no pain or griping. The diarrhœa often recurs again and again, and after having been as often checked by remedies, clears away any œdema which may remain, gives a pinched and sunken aspect to the patient, and eventually carries him to his grave. When this has been the case it is found that the small intestine has been affected, so that the vessels of the mucous membrane give the iodine reaction. Vomiting similarly indicates the change in the vessels of the stomach, a change which allows a morbid exudation to take place into the cavity of the organ. It often begins with loss of appetite and nausea after food.

As a counterpoise of these dangers it is found that patients suffering with this disease are but little liable to the affections of the brain which have been described as uræmic. Such attacks are the exception, not—as with other forms of renal disease—the rule. Convulsive seizures are more common than simple coma. In a series of 48 cases, all with marked symptoms of this renal disease, and all exhibiting it on *post-mortem* examination, there occurred 3 instances of uræmic convulsions, 2 of simple coma, 1 of unnatural drowsiness. This is a proportion of nervous disturbance infinitely below what occurs in albuminuria from other causes.

The freedom from these affections probably depends upon the fact that the constitutents of the urine are much less diminished than in other diseases of the kidney, while death is often due to the progress of the disease in the bowels.

The most frequent cause of death in this disease is diarrhœa; the next pneumonia. Inflammation of the serous membranes, though very common, seldom causes death, unless the membrane affected be the peritoneum. Looking at the cause of death, as far as it could be isolated, in 35 cases in which that result was apparently due to the renal disorder, it was found that

Diarrhœa	was the chief cause of	death in	13	cases.
Pneumonia	"	"	7	"
Peritonitis	"	"	4	"
Convulsions or coma	"	"	4	"
Pleurisy (one associated with erysipelas)			2	"
Ascites (liver affected and tapping performed)			1	case.

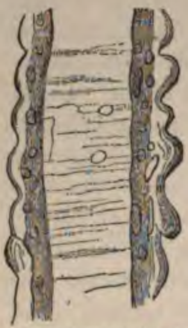
Excessive general dropsy, vomiting, bronchitis, enteritis, and coagulation of blood in the pulmonary artery, each contributed one death; while pericarditis and erysipelas, though apparently not the chief cause of death in any case, were present in some instances.

Thus it appears that the tendency to death is by inflammation of the lungs and serous membranes, or by exhausting diarrhœa. In the frequency of the latter complication and the comparative immunity from head symptoms the disorder differs from other forms of renal disease. It resembles them in the liability to inflammatory attacks.

The state of the circulatory system with lardaceous disease of the kidney is of interest not only in relation to the disorder itself, but as throwing light upon the process by which in renal disease of other kinds hypertrophy of the heart and various changes in the vessels are brought about. Lardaceous disease uncomplicated by secondary renal changes probably has no effect in causing cardiac hypertrophy; and it is to be observed at the same time that neither does it occasion increased arterial tension nor, as a rule, any uræmic symptom. But it has been shown that sometimes both marked renal fibrosis and also tubal inflammation may ensue upon the lardaceous infiltration, and with these, as might be anticipated, various uræmic consequences, as displayed upon both the nervous and vascular system, ensue. Thus, occasionally the thickening of heart and vessels with retinal hemorrhage and the whole cardio-vascular series may be traced to lardaceous disease. The infrequency with which these results occur in connection with this disorder is no doubt due to the small impairment of renal secreting power which takes place until the disorder is advanced and complicated; and also, as Dr. Sibson has suggested, to the frequent persistence of exhausting discharges, which, whether as suppuration, which has caused the disease, or diarrhœa and vomiting caused by it, relieve the blood-vessels and keep down their tension.

The general absence of hypertrophy of the heart with lardaceous disease has been remarked by all recent writers on the subject. The annexed table (p. 195), which was published in the first edition, in 1867, gives a proportion of 2 per cent. Dr. Grainger Stewart, in the following year, gave a proportion of 4 per cent. Dr. Galabin in 1873 found hypertrophy in 3 of 15 cases; Dr. Sibson also 3 in 15. As the latest contribution to this question I may state that in the series of 83 cases of lardaceous disease referred to at page 177 which relate to the disorder in general, not as affecting the kidneys especially—though few cases are comprised in which these organs were not affected—the heart was noted as hypertrophied in 6, while in 3 it was smaller than natural. These estimations, of which the later take notice of the smaller degrees of enlargement which probably in the earlier passed without mention, agree in representing hypertrophy of the left ventricle as only exceptionally associated with the disease under consideration.

With regard to the arteries these have already been alluded to as the early and favorite seats of the peculiar infiltration. Whether these be examined in the pia mater, in the kidney, or elsewhere, the smaller of these, together with certain groups of capillaries, such as belong to the malpighian body, became permeated throughout their substance with the translucent iodine-loving material. The muscular coat becomes variously altered, its transverse striæ curiously exaggerated, and the outer coat swollen, as if distended somewhat irregularly by the deposit. The muscular coat, however, does not appear to become actually thickened, save with those exceptional cases in which the disorder is associated with cardiac hypertrophy and the other accompaniments of chronic uræmia. It is of interest to observe that, however the coats of the systemic vessels may be swollen by the lardaceous deposit, yet the ventricle will usually remain without enlargement, unless an uræmic hindrance be superadded.


(In glycerine) 375 diameters.

The infrequency of vascular and cardiac hypertrophy with lardaceous disease may be taken as an index of the rarity in the same circumstances of the various hemorrhagic concomitants of the cardio-vascular change, such as cerebral and retinal extravasation. Cerebral hemorrhage is exceedingly rare with the lardaceous change. I have not myself witnessed an instance, though some have been referred to by other writers. With regard to retinal hemorrhage an example, in which this condition in a well-marked shape coexisted with advanced lardaceous disease, came under my own notice, and the same concurrence has been noticed, though as one of much infrequency, by other writers (see page 248). Thus, lardaceous disease, subject as it is to be followed by catarrhal and fibrotic changes, may be ultimately attended by the disorders of circulation and of the circulatory system which especially belong to these secondary conditions.

Hemorrhagic accidents other than have been referred to sometimes happen with advanced lardaceous disease, perhaps in double connection with arterial deterioration and the attenuation of blood which is a concomitant of the exhausting lesions which so often belong to the complaint. Epistaxis is the most frequent of these, but is, perhaps, less so with the lardaceous than with the granular disorder. Purpura is a more uncommon manifestation of the same tendency ; and as one of still greater infrequency—I have only found one instance—must be mentioned intestinal hemorrhage.

In connection with the state of the circulating system under the influence of this disorder it must be stated that endocarditis, or, to speak more precisely, the deposition of ragged fibrinous matter upon the valves, and consequent embolism, are comparatively frequent. Both vegetations and blocks have been known to give the lardaceous reaction. The matter deposited within the heart is probably connected in its origin with that which infiltrates the organs.

In the 83 cases of lardaceous disease referred to at page 177, recent vegetations were found on the valves in 5 cases, valvular thickening of old date in 21, a proportion of valvular mischief which belongs to no other renal affection.

The following table gives an abstract of the symptoms observed in 48 cases in which the disease arose independently of any other renal affection:

Table showing Symptoms and Results of Lardaceous Disease of the Kidney, derived from Analysis of 48 *Cases under the Observation of the Author, in which* Post-mortem *Examinations were made. Those Cases only are included in which the Disease had Progressed far enough to cause Albuminuria and other Symptoms Referable to the Kidneys.*

Hæmaturia (blood evident without microscope)	4
Frequency of micturition	4
Pain in loins	5
Œdema	33
Ascites	12
Hydrothorax	1
Fluid in pericardium	0
Erysipelas, or inflammation of cellular tissue	3
Uræmic convulsions	3
Simple coma, or semicoma	2
Other head symptoms	1
Pneumonia	9
Pleurisy (present in a recent state at time of death)	5
Peritonitis " " " "	4
Pericarditis " " " "	3
Endocarditis " " " "	1
Bronchitis " " " "	1
Vomiting (not of blood)	11
Diarrhœa (motions in one case bloody)	22
Enteritis	1
Amaurosis, or dimness of sight	0
Sanguineous apoplexy	0
Epistaxis	2
Purpura	2
Vomiting of blood	0
Hypertrophy of heart (simple)	1
Atheroma of arteries	1
Gout	2

URINE.

The urine in this disorder resembles in many particulars that which belongs to granular degeneration. A general rule holds good, that the urine is diminished by obstruction of the tubes, increased by disease confined to the intertubular structures.

The first change in the urine in this disease is an increase of quantity. The amount varies from the normal average up to perhaps four times as much. Dr. Grainger Stewart places the maximum at 200 oz. in the twenty-four hours. I have never seen so much. The common range at the earlier periods of the disease is from 50 to 90 oz. The urine thus increased is pale, clear, and watery. It has a low specific gravity—1015 to 1006, or even less. When the increase becomes obvious, or soon afterward, a trace of albumen appears. Commencing always in minute quantity, it slowly increases until it is sufficient to form a bulky coagulum. Toward the later periods of the disease the secretion generally becomes reduced in quantity. This appears to depend either upon the presence of a certain amount of tubal catarrh or upon a large proportion of the tubes being stopped up by the fibrinous exudation. Upon whatever cause it may depend, the occurrence is very general in the later periods of the disease. The urine falls to its natural quantity, and often below. Sometimes it is reduced to 8 or 10 oz. in the twenty-four hours, but this is uncommon ; it comparatively seldom falls below 20. The extreme diminution which is characteristic of nephritis does not take place. When the urine has become thus reduced in quantity it is generally highly albuminous.

The acidity of the urine is decreased in this as in other forms of albuminuria.

Blood is but rarely present, less often than with granular degeneration, much less often than with nephritis. In a series of 48 cases in which the urine was examined during life and the body after death, there were but 4 in which the secretion was obviously discolored with blood. In all these, however, the hemorrhage was considerable ; in one, constant and profuse.

Casts make their appearance at a very early period, while the albumen may be yet a mere trace, and they increase in abundance as the disease progresses. Although there are none which are peculiar to this complaint, if we except those rarely found which give the iodine reaction, as shown in Plate XI., those which more especially belong to it are simple cylinders of fibrine. These may be large or small, according to circumstances, and may be frequently dotted with oil, as the consequence of a change within themselves. Such casts are almost always present in this disorder, though they are not confined to it. They are generally of small or moderate diameter, evidently formed in tubes which retain their epithelial lining ; sometimes their larger size and stouter make shows them to have proceeded from tubes either denuded or much dilated. Besides these, casts sometimes occur which contain, or chiefly consist of, epithelial cells, and when these are present loose cells of the same nature often abound. In such cases (Carter, p. 209,) a catarrh of the tubes has been superadded to the primary disease. The engraving (Plate VII., Fig. 2) includes all the varieties of casts which were found in the cases upon which this account of the disease is chiefly based.

It is by no means rare, when the urine is scanty, to find a deposit of uric acid or urate of soda.

PLATE XI., *to face page* 196.

Fig. 1.

Section of a Lardaceous Kidney, made after freezing, with the tubes occupied by casts which give the lardaceous reaction with iodine. Magnified 75 diameters.

The section was made from the kidney of Roberts, to which Fig. 2, Plate X., also relates. The figure is reproduced from the "Medico-Chirurgical Transactions for 1867."

Fig. 2.

Shows the iodine reaction of dealkalized fibrine, which is similar to that of lardaceous tissue.

Some masses of fibrine as obtained from the heart, and portions of the substance obtained by dissolving the same in dilute hydrochloric acid, and recovering by evaporation, as explained at page 174, have been exposed to the action of the same solution of iodine and the results portrayed.

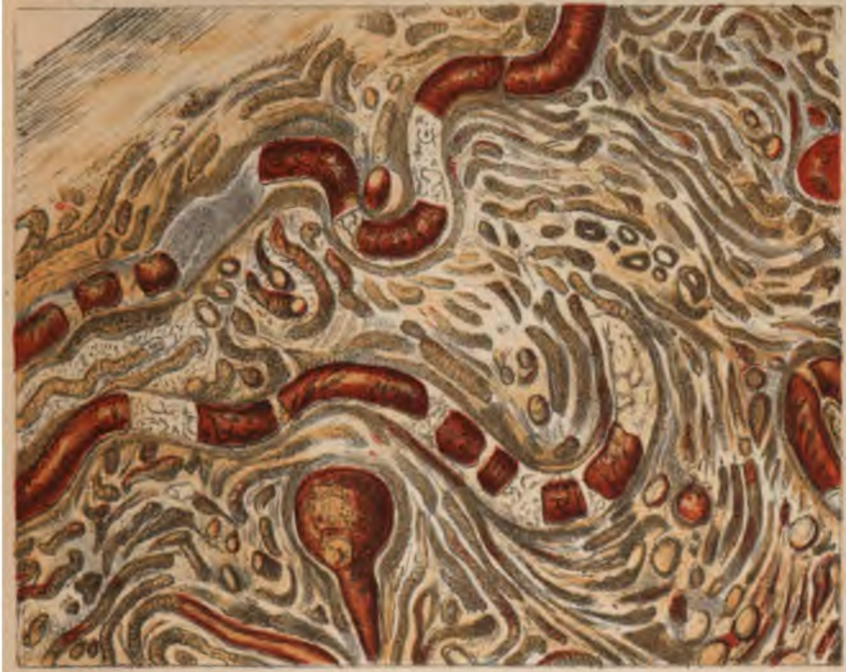


Fig. 1

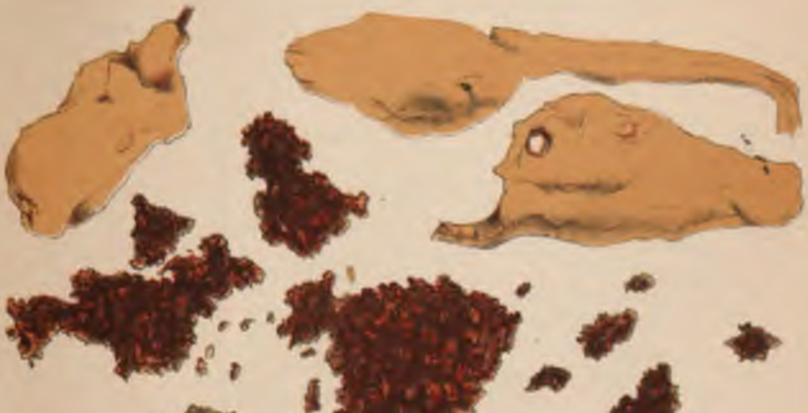


Fig. 2

CHEMICAL CHARACTERS OF THE URINE.

While the urine exceeds its proper amount—that is, during the greater part of the disease—the urea falls but little below the normal amount. Toward the end, when the urine is scanty, this important constituent is more sparingly excreted, but never approaches the degree of diminution which results from the other forms of renal disease.

Among many cases in which I have ascertained the daily amount I have never chanced to find the quantity below 7 grammes. The ordinary range is from half to two-thirds the normal quantity. In 7 cases in which the urea was estimated it varied from 7.35 to 24.9 grammes.

A case is related by Rosenstein,[1] in which the urine became reduced to an unusually small quantity, and the urea fell as low as 3.6 grammes in the twenty-four hours ; but this must be regarded as an unusual circumstance. It may be stated as a general rule, that so long as the urine exceeds, or does not fall short of, the proper quantity, the urea will not be greatly diminished.

The uric acid is sometimes normal in quantity, more often diminished, occasionally absent. In two cases under my own care in the hospital I was unable to discover any.

The phosphoric acid is always reduced—diminished with constancy in well-marked cases of the disease to a sixth or even a smaller proportion of its proper amount. The reduction of this constituent appears to be more regular than with either of the other forms of renal disease, though it seldom reaches their minimum.

The sulphuric acid is less reduced than the phosphoric. It is nearly always more abundant than that acid, though in health it exists in smaller quantity. This is much what occurs with granular degeneration.

It appears that the chlorine, though reduced to a less extent than occurs in nephritis, is diminished more than, as a rule, occurs with granular degeneration. It is perhaps not necessary to give a detailed account of the analyses which have been made, as the cases reported will bear out the statements with regard to this and the other constituents of the urine.

The alkaline salts in this disease appear to be below the normal amount. In the cases of King (p. 205) and Gilbert (220) both potash and soda were greatly reduced, especially the latter. In these cases a purulent discharge was flowing at the time of the examination. How far these alkalies are lessened when this renal disease is present under other circumstances, and what proportion they bear to their amount in other forms of albuminuria, are questions as yet unanswered.

Nothing remains to be added to what has been said about albumen. It is less abundant in this disease than in nephritis; more, as a rule, than with granular degeneration.

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[1] Frau Meller, p. 246.

Summing up the chemical changes which occur in the urine as a consequence of the lardaceous change in the kidney, they are as follows:

The water is increased, except in cases of long standing, when it is frequently diminished. With this exception, all the elements of the urine are diminished. The urea is reduced, but not to the extent which occurs with other forms of renal disease.

The amount of uric acid is variable.

Of the mineral constituents that which is diminished with the greatest regularity is the phosphoric acid.

The following cases, together with those placed after the section upon treatment, illustrate the pathology and symptoms of the disease.

*Dilatation of Bronchial Tubes, with Profuse Suppuration—Amount of Alkali lost Daily—Symptoms of Lardaceous Disease: Albuminuria, Dropsy, Diarrhœa—Treatment by Tonics and Salts of Potash—Death—*Post-mortem *Examination.*

Fanny White, aged six years, became my patient in the Hospital for Sick Children, Jan. 29, 1866.

She had had cough since birth. At the age of two years she had a severe attack of whooping-cough, after which the former symptoms continued, with gradually increasing expectoration. For the last two years she had been easily put out of breath, and the least excitement caused much cough. For six or eight months she had been pallid and emaciated, and for a month the feet had swelled. The urine on examination was found to be albuminous, and she was thereupon admitted.

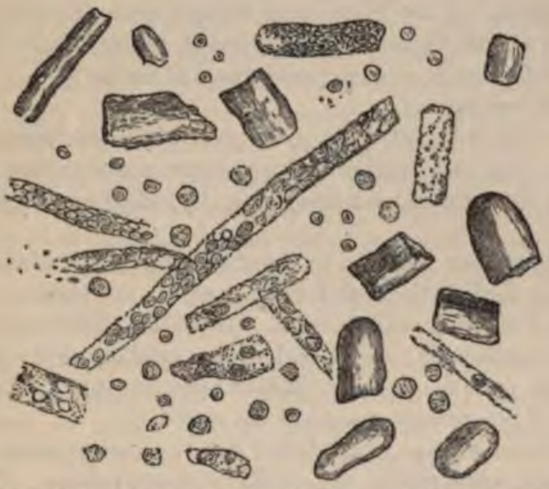
She was pale and puffy; the legs were œdematous. There was much cough, attended with copious purulent expectoration, which had an odor suggestive of gangrene. There was slight general dulness over both lungs, which gradually increased from above downward. The apices were nearly natural on percussion; it was thought that the right was rather the less resonant. The dulness over the lower lobes was very decided. Large bubbling and cavernous sounds were heard all over the chest, especially low down. The voice sounds were slightly increased. From these signs, as well as from the general symptoms, it was thought that the lungs were excavated by vomicæ, the lower lobes more than the upper.

The pulse was quick, the appetite bad, the bowels regular.

The urine was repeatedly examined: it was always scanty, varying from 350 to 500 c.c. in the twenty-four hours. The specific gravity varied from 1014 to 1023. It was highly albuminous, sometimes turning nearly solid when boiled. It threw down, very generally, crystals of uric acid. Under the microscope abundant casts were seen, mostly of considerable diameter and uniform waxy character. Besides these other casts were found which contained blood globules and epithelial cells. Loose cells of renal epithelium were scattered about, some of which approached pus globules in character.

The purulent discharge, which was regarded as the origin of the mischief, was examined. It was found that a quantity not varying much from 105 c.c., or about 3 oz., was spat up every day. It was alkaline; it had the appearance of unmixed pus; the odor was offensive. This contained .932 of ash, .877 soluble in water, .055 insoluble. The soluble ash contained of potash .089, soda .275. These numbers represent the amounts contained in twenty-four hours' expectoration.

The child was put upon liberal diet, with wine. She had cod-liver oil with reduced iron; and as a means of compensating for the loss by the discharge, she was ordered a mixture containing citrate of potass, which was associated with cinchona and squills. She went on well for a time, but had frequent attacks of diarrhœa, which were supposed to depend upon the extension of the disease to the bowels. These were held in check for a time by the use of injections of starch and laudanum, and subsequently by astringent medicines, of which sulphate of copper and opium were found to be most effectual. By March 2d the disease had evidently made progress; the child had perceptibly wasted and become weaker; the expectoration was more profuse and offensive. Two days later she was taken out by her parents, the same treatment being pursued while she was at home. She gradually sank, and died on the 11th.



Urinary deposit, Fanny White. Loose renal epithelium. Hyaline and epithelial casts.

Permission was obtained to examine the body. It was much emaciated.

The pleural cavities were closed by old adhesions. The upper lobes of both lungs were emphysematous, but otherwise natural. The lower lobes were almost entirely occupied by large globular or oval cavities, which varied in size from a walnut downward, most of them approaching the size mentioned. They proved to be simple dilatations of the bronchial tubes. These, where they entered the lower lobes, dilated into large cavities, each like a flask, with a narrow neck and a large body. Tracing down any one of the large bronchi, it was found to divide into smaller tubes which terminated in oval cavities such as have been described. These were lined with a mucous membrane continuous with that belonging to the bronchial tubes, and resembling it closely. These cavities were not surrounded by any consolidation such as usually abounds in the neighborhood of vomicæ; their walls were no stiffer than ordinary mucous membrane. In many places the walls of the cavities were in contact with those adjoining. The bronchial membrane was generally injected, and the dilatations were loaded with pus. A small part of the lower lobe of the left lung was occupied by ordinary red hepatization. A few places were noticed near the thin edges of the lobes, where a little fibroid tissue separated the cavities. There was not a tubercle in the lungs or in any other part of the body.

The heart was natural.

The liver was enlarged, fatty, and gave slight iodine reaction.

Both the large and small intestine were extensively altered in the same manner, the characteristic test being given by iodine in a very marked manner.

The kidneys were enlarged to about the size proper to the adult. The capsules were slightly thickened and adherent; the surfaces were smooth, very pale, but showing stellate veins. On section there was a general look of unnatural uniformity; cortex and cones partaking of the same pale buff color. Iodine gave the peculiar reaction upon the malpighian bodies, and upon many of the straight vessels of the cones.

Under the microscope it was found that the cortical tubes were in some instances open, while others were stuffed with epithelium. The cells taken separately were quite natural. The malpighian bodies were enlarged and their capsules were dilated into globular cavities.

This case is interesting in several respects. The primary disease, dilatation of the bronchial tubes, is one of rare occurrence, and one which is usually mistaken, as in the present case, for phthisis. The complete absence of tubercle, however, shows that the secondary disorder did not depend upon any scrofulous cachexia. The daily discharge of pus was very great, and at once led to a suspicion as to the nature of the renal disease, which was converted into a certainty when the waxy casts were found, and diarrhœa became a prominent symptom. The fact that besides simple fibrinous casts, casts were found of the epithelial variety, besides scattered cells of renal epithelium, more or less puriform in character, showed that, as is often the case, a certain amount of tubal inflammation had been superadded to the original change. With this the urine became scanty. After death many of the tubes were found obstructed by epithelium. The treatment which was adopted was warranted by experience, so far as the liberal diet, cod-liver oil and iron, were concerned. The citrate of potass was given in order to compensate for the loss in the discharge. Like most cases of this variety of renal disease, the patient sank worn out by the primary disease and by diarrhœa, never having had any cerebral or proper uræmic symptoms.

Enormous Enlargement of the Liver and Spleen, together with Lardaceous Disease of the Kidneys and Small Intestine, consequent upon Long-standing Disease of the Vertebral Column—Death from Erpsipelas— Post-mortem.

A small, extravagantly misshapen child, named Laura Argent, whose stature was about that of eight, while her years were fifteen, but whose wizened and aged countenance corresponded ill with her small frame, became my patient in St. George's Hospital in October of 1873.

Her condition was typical in many respects of an extreme degree and advanced stage of lardaceous disease. The spine was distorted by an angular curvature which caused the pelvis to stand out abruptly behind the vertebral line; several sinuses surrounded by irregular cicatrization opened upon the root of the neck behind the left sterno-mastoid muscle; the face, thorax, and limbs were wasted to skin and bone, while the abdomen presented a tight and globular distention at least equivalent to that of concluding gestation.

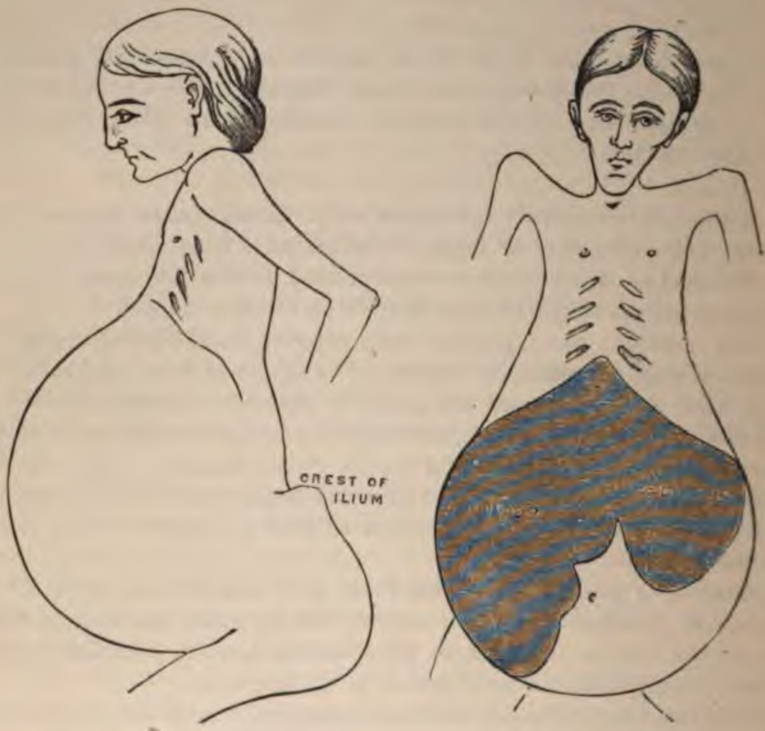
Examining into the abdominal swelling, it appeared to be wholly due to visceral enlargement; a firm smooth surface, which was thought to belong to the liver on the right, to the spleen on the left, occupied as much of the abdominal district as is shaded in the diagram.

The mass, the prominence of which is represented in the side view, reached on the right side from the edge of the thorax to the line of the groin, on the left from the edge of the thorax to the level of the umbilicus. The swelling in the right side reached 12 inches below the ribs in the nipple line; on the left side the corresponding measurement was 5½ inches. A deep notch vertically above the navel partially divided the mass into two great segments; the division, however, was not to be traced above the notch by any fissure or furrow, and it was not possible to determine whether the notch marked the division between the right and left lobes of the liver, or between the liver and spleen. The former proved to be the case.

No fluid could be detected in the abdomen, nor was there œdema anywhere. The urine was albuminous.

The visceral enlargement, affecting, as it appeared to do, both the liver and spleen, taken together with the open sinuses and spinal curvature, was enough to declare the nature of the disorder without further question.

Inquiring into the antecedents, it was found that she had been the subject of purulent discharge from the age of three years, when the sinuses at the root of the neck opened, and at the same time the spine began to show abnormal curvature. The discharge continued and increased, so that for the last two years it amounted to about a quarter of a pint daily. The abdominal enlargement was first noticed when she was seven years old, coincidently with an attack of dysentery, for which she was under treatment in the Colchester Hospital. During the last two years the swelling had greatly increased.



She had hitherto had no obvious sign of lardaceous disease beyond the enlargement of the viscera and slight œdema of the legs; no diarrhœa or vomiting, nor much complaint of thirst. She had had frequent attacks of somewhat profuse bleeding at the nose.

She was put upon liberal diet and tonic treatment, comprising iron, quinine, and cinchona; while she had in addition small doses of liquor potassæ with citrate and phosphate of potash, but it was evident from the first that she had come in only to die, and her downward progress was never materially interrupted.

Not to dwell in daily detail upon her decline, the œdema reappeared, fluid became evident in the abdomen, giving fluctuation in the regions unoccupied by the swollen viscera, and she became affected with dyspnœa, accompanied with dulness, tubular breathing, and scattered moist sounds over as much of each lung as was below the scapula. Later, diarrhœa became a prominent symptom and vomiting an occasional one. The special treatment was often interrupted as individual symptoms had to be met; it was never urged to the extent of making the urine alkaline. Ultimately she was attacked with erysipelas of the face, which reappeared in a second situation after apparent recovery, and was the immediate cause of her death when she had been three months in the hospital.

On examination of the body after death the unusual diminution of the the thoracic cavity was conspicuous; it was narrowed laterally, with much thrusting forward of the sternum, while the abdomen had encroached upon it from below to a very unusual extent.

The lungs were congested and compressed, but otherwise natural.

The heart was natural.

The abdominal cavity contained much fluid and many old bands of adhesion between the liver and the spleen, which organs had been flattened by mutual pressure so as to present unbroken continuity of surface. The notch above the umbilicus marked the separation of the right and left lobes of the liver, the spleen having been thrust to the left by the left lobe of the liver, to which its lateral surface was so exactly adapted that no superficial inequality marked the distinction. The liver weighed 86 oz.; it presented the lardaceous reaction in an extreme degree. The spleen was enlarged to the weight of 14 oz. In section it was pale and waxy, and gave the iodine reaction characteristically. The kidneys weighed together 6 oz., their surfaces were uneven, their substance generally pale, and the special reaction strongly and widely displayed upon the application of iodine. The small bowel likewise gave the lardaceous reaction.

The fourth, fifth, and sixth dorsal vertebræ were carious, and were surrounded by a collection of thick pus which reached to the diaphragm.

This narrative is inserted as bearing upon the general aspects of lardaceous disease rather than upon the kidneys in particular. The case is one of many in which a similar suppurative process, continued in this instance for twelve of her fifteen years of life, has led to a like result. The hepatic enlargement was the most striking feature in the case, and the extent of this organ over the whole abdominal front, as exposed after death, was remarkable. The spleen, inseparable by touch from the liver, was pushed, notwithstanding its bulk, quite into the left hypochondrium, where the greater part of its bulk lay behind the left lobe of the liver. The uræmic results of the renal disease were entirely absent. The dyspnœa of the close was mainly due to the encroachment of the abdominal upon the thoracic cavity, and consequent embarrassment of the lungs. It is to be noted that no tubercle was present. The erysipelas which was the immediate cause of death was possibly due to the infective or imperfectly pyæmic results of the original suppurative lesion.

General Lardaceous Infiltration following Syphilis and Dysentery Contracted in India—Post-mortem *Examination—Analysis of Liver—Complicated Influences Concerned in the Origin of the Disorder.*

James Barry, a discharged soldier, forty-five years of age, who had served fourteen years in Bengal, and suffered from fever, ague, syphilis, and dysentery, was frequently a patient at St. George's Hospital, alternately under Dr. Fuller and myself. He had a bronzed complexion and sharp features. He was emaciated, while the legs were very œdematous. The liver projected much below the ribs. This, with the characteristic appearance of the man, and the fact that his urine was loaded with albumen, led at once to the belief that he was the subject of lardaceous infiltration. He suffered much from vomiting and diarrhœa, became more wasted and prostrate, and finally sank, after having had a protracted attack of epistaxis.

At the post-mortem examination there was found a scar upon the penis, such as would result from a chancre.

There was a cicatrix at the apex of the left lung, with puckering of the surface and evident loss of lung-tissue. Some quiescent tubercle occupied one of the bronchial glands.

The liver was tightly adherent to the diaphragm by old false membrane. The capsule was thickened, and presented on its surface a number of little pits or depressions. The organ was greatly increased in size, and on section presented a most characteristic "waxy" aspect. Iodine produced the characteristic tint over a large proportion of the surface. In the centre of the right lobe was an irregular firm cream-colored mass, as large as an orange, harder and tougher than tubercle, which under the microscope showed traces of a fibroid structure, and was presumed to be a syphilitic deposit.

The spleen and kidneys were also lardaceous, as was evident by their appearance and by the reaction of iodine. The intestines were in the same state throughout their entire course; they were free from ulcers and scars.

A most careful examination of the body failed to show any bone disease, or any appearance which could be interpreted as a certain record of extensive suppuration.

The liver, so extensively occupied by the waxy deposit, was examined in further detail. This was done with more than ordinary care, in consequence of the uncertainty which involved the origin of the disease.

The cut surface gave, with litmus, a decidedly acid reaction, which was imparted to water or spirit in which pieces of the organ were soaked.

The acid thus extracted proved to be non-volatile. The acid solutions, obtained as described, contained a large quantity of phosphoric as compared to the other mineral acids, and it was surmised that the reaction was due to the presence of acid phosphates. Whether due to this or to free acid, it appeared that the excess was rather relative than absolute, depending apparently upon deficiency of alkali rather than superabundance of acid. 100 grammes of the fresh liver were reduced to an ash, and the alkaline salts extracted in the usual manner. These amounted to .633 grammes, the average of health being 1.00. An accident prevented the separate estimations of the potash and soda. The diminution in the mixed alkaline salts is quite enough to account for the unnatural acidity of the tissue. The earthy salts amounted to .154 grammes, the normal average being .057. This increase accords with what is generally found under the circumstances.

Both syphilis and dysentery entered into the antecedents of this case; it is probable that the former was mainly concerned in its causation. As with many of the most extreme instances of the disorder, it was of tropical origin. An example of the disease in a very similar shape, also of Indian source, and involving in its rise the combined influences of syphilis and dysentery, is to be found at page 217, as an instance of successful treatment. It is probable that in each instance the lardaceous effect of the constitutional syphilis was enhanced by that of the dysenteric discharge; while the exhausting action of climate lessened the power of resistance which a state of vigorous health and ample nutrition would have presented to both.

The following cases not only illustrate the general pathology and symptoms of the disease, but have interest in regard to questions of operative surgery. Two of the patients were indeed made the subjects of excision of the knee.

*Strumous Disease of Wrist, with Profuse Suppuration—Phthisis—Urine Increased and Albuminous—Analysis of Urine—Thirst—Œdema—Diarrhœa—Death from Exhaustion—*Post-mortem *Examination—Estimation of Potash and Soda in Liver.*

On October 1, 1866, I was asked by Mr. H. Lee to examine one of his hospital patients with regard to the medical aspects of a proposed operation.

The patient was a laborer, named John King, twenty-six years of age. According to his own account he had, seven months previously, sprained his right wrist by carrying a weight; a few days afterward the joint became painful, and two months later the disease had progressed so far that a sinus had formed which discharged pus. The purulent discharge became very profuse, amounting, according to the man's own account, to a pint a day; an estimate which was exactly confirmed by the nurse of the ward, who put it down at 20 oz. A month after the abscess broke he began to cough, the cough after a time being accompanied with purulent expectoration. Within the last two months the urine had increased in quantity and in frequency of passing; he suffered from thirst—"drouth," as he called it—and the bowels became loose. Latterly the legs became œdematous, the urine still remaining in excess.

When I saw him he was much sunk and wasted. There was much soft œdema about the ankles; no enlargement could be felt of the liver or spleen. He had a cough, with muco-purulent expectoration; and on listening at the chest, dulness, increased voice sounds, and large bubbling, were heard at the right apex. There was not much thirst, the tongue was red and raw-looking, the pulse 108. There was extensive disease of the bones belonging to the right hand and forearm, with numerous openings, which discharged now about a quarter of a pint of pus daily.

The urine was collected for the twenty-four hours ending October 1. It was acid, slightly turbid.

Quantity	=	2040 c.c. (or 72 oz.)	
Sp. gr.	=	1007	
Urea	=	22.4	grammes.
Uric acid	=	0.0	"
Phosphoric acid	=	.51	"
Sulphuric acid	=	.969	"
Chlorine	=	.948	"
Soluble ash	=	4.263	"
Insoluble ash	=	.265	"
Potash	=	1.453	"
Soda	=	.723	"

Under the microscope many slender casts were seen, such as are represented in Plate VII, Fig. 2. These were simple cylinders of fibrine, more or less dotted with specks of oil. They were treated with a solution of iodine, but did not become more deeply colored than the scales of epithelium and other objects which came in the way of the fluid.

The opinion which was given was to the effect that the kidneys and the bowels were lardaceous, and that in addition the right lung was the seat of advanced tubercular disease. Any serious operation was of course out of the question.

From this time, in spite of the most liberal and varied diet, he gradually sank. The expectoration continued in considerable quantity, but the discharge from the wrist became less. The diarrhœa at last was less frequent. The urine remained copious, while the œdema and thirst persisted. On October 13th he died, without any further symptoms.

At the *post-mortem* examination it was found that all the carpal bones, the ends of the metacarpal bones, and of the ulna and radius, were bathed in pus, and were soft enough to be cut with a knife. The ulna protruded from a wound at the back of the wrist.

Both lungs contained much crude tubercle, and at both apices were vomicæ, that on the right side being large and ragged.

The liver was of about the natural size; it was pale, and gave with iodine the characteristic reaction. This reaction took place in a decided manner, but to a comparatively small extent. The spleen was natural in size and appearance, but with iodine became closely dotted over with very characteristic brown specks.

The kidneys were increased in size ; one weighed 7½ oz., the other about the same. The capsules were slightly adherent, the surfaces mottled with vascularity. The cortex was increased, generally white and coarse in texture. With iodine the malpighian bodies were brought out in strong relief, looking like grains of brown sand sprinkled upon the surface.

Under the microscope it was found that the epithelium was very fatty, as is often the case when the lardaceous change has taken place.

The small intestine was colored in the same characteristic manner. In both the large and small bowel were several small, clean cut ulcers, such as result from tubercular disease.

Subsequently 100 grammes of the liver were reduced to an ash, and the potash and soda estimated. The following are the results. The healthy averages are annexed for comparison ; the amounts are given in grammes.

	John King.	Average of Health.
Soluble ash	.481	1.00
Insoluble ash	.209	.181
Potash	.12	.169
Soda	.053	.156

This case is a good example of the class to which it belongs. The disease arose from suppuration, which, though it had continued for a shorter time than is generally the case, was in unusually large amount. The sinuses opened only five months before the death of the patient, but there was evidence to show that for a time as much as a pint of pus had been discharged daily.

The symptoms were as characteristic as the history. The urine albuminous and increased in quantity; notwithstanding the excess, œdema; the patient reduced by a watery flux from the bowels, sinking at last without any cerebral disturbance. These facts, even without the history of suppuration, would suffice to make the diagnosis almost a matter of certainty. The abundance and character of the fibrinous casts, and the fair proportion of urea which still passed, are also distinctive.

The circumstance in particular, that œdema increases while the water is in excess, is, if not peculiar to, at least very suggestive of, this form of albuminuria. It is probable that the effusion depends upon the extension of the disease to the blood-vessels of the cellular tissue, as well as upon, as in other forms of renal disease, the impoverishment of blood. The deteriorated vessels allow the serum to transude. The thirst which is present in such cases is probably consequent upon the multiplied demand for fluid which is passing off by diuresis, from the bowels, and into the cellular tissue. The deficiency of the alkalies in the liver was extreme, although on the application of iodine the brown color did not come out over so large a proportion of the section as is sometimes the case. The discharge continued up to the time of death, and it is probable that with so large a drain, the proportion of alkali suffered not only by the deposition of the specific dealkalized matter, but by the removal of all spare alkali from the blood and tissues. Where the discharge has ceased, or has much diminished, before death, so that the tissues are enabled to recover their ordinary condition, alkali is wanting only in the new material.

It may be believed that in cases like this one, where a profuse purulent discharge exists, the potash and soda of the urine suffer diminution. In health the daily amount of potash varies, according to Dr. Parkes, from 1.7 to 7.6 grammes. In this case the amount was below the minimum. Soda, which in health exists in larger quantity than potash, was here in smaller. Observations, however, both in health and disease, are too few to give safe conclusions.

Disease of Knee-joint, with Discharge of Pus—Resection—Sudden Death after the Operation—Disease of Kidneys not Discovered during Life—Coagula in Heart.

Emma Denman, aged nineteen, had disease of the knee-joint for the last ten years of her life. For the last fourteen months pus had been discharged from openings which had formed in the vicinity. She became a patient in St. George's Hospital, and the ends of the bones were excised. On the sixth day after the operation, the wound at this time looking well, "she suddenly became faint and collapsed, and died in a few minutes."

The *post-mortem* examination was made by myself. The body was fat. The ends of the bones were coated with a thin layer of lymph, and the wound had a healthy appearance. Every part of the body was examined, but nothing was found to remark excepting the kidneys and the contents of certain blood-vessels. The kidneys were enlarged, their surfaces were roughened, and their capsules adherent. Their substance was of a pale fawn-color, an unnatural whiteness being disguised by congestion. The application of iodine brought out the characteristic "amyloid" reaction on the malpighian bodies, the vessels of the cones, and also on some of the vessels of the cortex. The change was well marked, and must have been the result of disease of some standing. No such alteration was found elsewhere, though all the likely places were put to the same test.

The right auricle was distended with a mass of elastic semitransparent fibrine. The other cavities of the heart contained similar fibrine, but in smaller quantities. The pulmonary arteries were occupied, but not distended, with soft black coagulum. The lungs themselves were natural. There was a small cylinder of buff-colored fibrine lying in the left middle cerebral artery, close to its origin. This was such as to fill the vessel without distending it. The aorta and all the large vessels were free, as were all the other cerebral vessels.

The viscera, and every part of the body excepting those mentioned, were perfectly natural.

Though the urine was not examined during life, there can be no doubt that it was albuminous. It is sufficiently evident that the kidneys had been long diseased. The suddenness and manner of death were such as would result from coagulation of blood in the cavities of the heart. The large mass of fibrine in the auricle was such as could not collect during the act of death, for death took place too rapidly to allow of the separation of fibrine to any considerable extent. The fibrine was deposited during life, and death occurred in consequence of the obstruction so produced. The inference that the fibrine had accumulated during life is borne out by the fact that a plug of the same material had been propelled into one of the cerebral arteries. The existence of albuminuria is known always to increase the proportion of fibrine in the blood, and in this case the protracted purulent discharge must necessarily have had an action of the same sort. The case furnishes an example of the tendency to formation of fibrinous coagula to which patients with the lardaceous disease are especially liable, though in every case of albuminuria there is more or less of the same proclivity. The operation, by weakening the powers of the circulation, appears to have acted as the exciting cause.

Abscess in Connection with Diseased Knee—Excision of Joint—Subsequent Lardaceous Disease of Kidneys Complicated with Nephritis—Post-mortem *Examination.*

At the beginning of the year 1867 I was asked by Mr. Pollock to see a patient, then under his care in the hospital, whom he believed to be affected by the "amyloid" disease. I saw her on January 3.

The patient was a girl, sixteen years old, named Charlotte Carter, of whom the following particulars were learned. Two and a half years ago an abscess had opened in connection with the left knee-joint, which for some time previously had been swollen. The discharge continued. Last April the ends of the bones were excised by Mr. Holmes, who had charge of the case in Mr. Pollock's absence. The wound remained partially open, and the suppuration had continued ever since. Latterly the urine increased in quantity. For three weeks there had been frequent action of the bowels, with watery motions. A week after the commencement of the diarrhœa the sound leg became œdematous.

When I saw her she was white and puffy. There was still a good deal of œdema, but less than there had been. No enlargement of the liver or spleen could be detected. The bowels were loose—seven times in the previous twenty-four hours. There was much thirst; the tongue was dry and red; the pulse 104, so feeble as to be hardly perceptible. The appetite was still fair, so that she was able to make use of the liberal and stimulating diet which was provided. Pain in the loins was present, and had lasted for several weeks. The urine was now very scanty. A little was obtained with difficulty, in consequence of the disturbed state of the bowels. It was deep colored and glutinous. It was loaded with albumen, so that it turned into a solid mass when boiled. There was not enough to give the specific gravity, or to allow of a chemical analysis. There was a very abundant sediment, of which the microscopic appearances are represented below. A great number of transparent waxy casts were present, many of large size. There was also granular casts, and others embedding epithelial cells. Besides the casts, there was also a most plentiful deposit of loose cells of renal epithelium.

Under these circumstances there was no room for doubt as to the condition of the patient. She was regarded as having the peculiar change in the kidneys which is associated with protracted suppuration, upon which a state of tubal catarrh had been recently superadded. This diagnosis had been scarcely written down when signs of prostration rapidly increased, and she quietly sank without any fresh symptom. A little time before death the diarrhœa ceased. There were no head-symptoms from first to last.

At the *post-mortem* examination the body was found to be fat and well nourished; the right leg œdematous.

The left knee was anchylosed, and there were several sinuses about the joint which led to softened and diseased bone; the end of the tibia proved also to be somewhat carious.

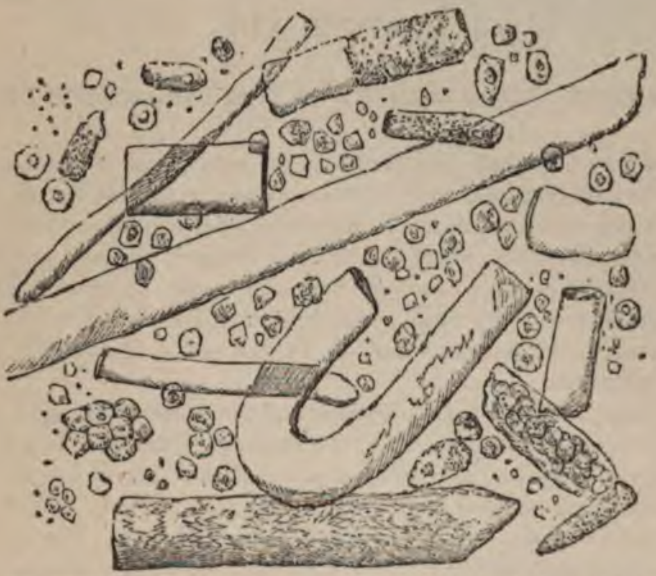
There was slight thickening of the mitral valve, and the aorta was slightly atheromatous.

The kidneys were greatly enlarged, together weighing 17½ oz. They were pale, but mottled with vascularity, the surfaces for the most part smooth and shining, though marked with a few small and incipient depressions. The cortex was very much increased, and loaded with an opaque yellow deposit, which had the appearance of being contained within a fine network of translucent gray material. In the right kidney were several fibrinous blocks, of old standing.

The malpighian bodies and lines on the cones gave a most marked iodine reaction.

With the microscope the epithelium was found to be perfectly natural.

Some tubes were natural, or only unnatural insomuch as the epithelium was held in position with more than natural tenacity. Other tubes were distended almost to bursting, with a granular packing, which abounded with epithelial cells and nuclei. Many were about four times their proper width, and were swollen into an irregular shape. A transparent section showed that numerous processes of fibroid tissue had formed upon the surface, and insinuated themselves between the tubes at somewhat irregular intervals. Within some of these were contracted remains of tubular structure.



Urinary deposit, Charlotte Carter. Loose renal epithelium, hyaline and epithelial casts.

It is difficult to look back upon this case without suspecting that, had the limb been removed when the joint was excised, the result might have been more fortunate. During the nine months which followed the operation the wound was discharging, and the visceral mischief necessarily extending. The kidney disease was, as often happens, latterly complicated with nephritis. It would seem that the new deposit acts as an irritant to the organ. A careful inquiry in this instance failed to elicit any external circumstance which could have led to the superadded attack. The tubal inflammation was as clearly recognized during life as after death. The pain in the loins, the scantiness and highly albuminous character of the urine, the intermixture of epithelial with the simply fibrinous casts, and the abundant discharge of loose epithelium, combined to place this beyond doubt.

CHAPTER XIII.

PREVENTION AND TREATMENT OF THE LARDACEOUS DISEASE.

Both prevention and cure lie within the capacity of the medical art in relation to lardaceous disease. To take prevention first, as the more attainable, our power in this respect is measured by the control which we have over the two great sources of the disease—suppuration and syphilis. To prevent syphilis is more desirable than easy; and our precautions as regards this source of the organic mischief must be limited to its early and efficient treatment, and the inhibition, by this means, of its remote and constitutional results.

As to suppuration, where an obvious and accessible discharge is in course of producing the complaint, the first consideration must be its arrest. The mischief will extend so long as the drain continues, but no longer. Whatever subsequent changes may ensue in the parts already involved, those which have escaped when the discharge ceases are safe. The morbid deposit, as we cannot but infer from *post-mortem* experience, must continually occur, in small quantity, in connection with transient suppuration, undeclared by symptoms, and probably itself harmless and transitory. With the cessation of the discharge the disease, far from extending, may possibly retreat toward health; in certain organs—the liver, the spleen, and the mucous membranes—there is irresistible evidence that it does so; while, at the worst, it will only prove progressive so far as secondary changes are actuated in the organs already involved. There will be no spread of the disease when once the suppuration has come to an end, and may even be much retrogression.

The extent to which recovery may take place under favorable circumstances, but without special treatment, was shown in the case of a boy now 13 years of age, who for half his life has been a patient of my colleague, Mr. T. Smith, and an *habitué* of the Hospital for Sick Children. Between six and seven years ago he began to discharge profusely from an abscess connected with a diseased pelvis and hip-joint. Within a year of this the liver and spleen began to enlarge, and continued to do so until four years ago the liver reached half-way to the umbilicus, the spleen to the crest of the ilium, the two together causing much abdominal prominence. The urine at the same time was slightly albuminous. Since this date, under appropriate surgery and frequent changes into the country and to the sea (the boy became the object of the persistent kindness of a benevolent lady, and thus had advantages which would not have commonly fallen to his lot), he has steadily mended, and now is in a condition, both as regards joint and viscera, which at one time appeared impossible. Not only has the discharge almost ceased, and the boy regained a fairly useful joint, upon which he can walk a couple of miles, but the liver no longer gives evidence of any enlargement; it is not to be felt below the ribs; the urine gives no trace of albumen; and the only relic of the lardaceous change which remains is in the size of the spleen, which still extends five inches below the ribs and to within two of the median line.

The means of arresting this disease at its birth, together with its parent suppuration, insomuch as they are chiefly surgical, can be here but generally and imperfectly indicated.

Where symptoms of the visceral change have become evident attempts to manufacture false joints, attended as they are by a tedious and exhausting process, will probably give way to the more simple and speedy relief of amputation. The question must often be reduced to a simple alternative between life and limb. In cases where, from the position of the diseased bone or any other circumstance, amputation is out of the question, the occurrence or the apprehension ot the change will supply a reason for removing the source of irritation by any other practicable means.

Persons with albuminuria from this cause have a considerable healing power, though they are exposed to greater dangers than commonly attend a surgical operation, and to some dangers—thrombotic, for example—different in kind from those which generally follow such a procedure. There can be no reason, on this ground, why a leg or an arm should not be removed, or why diseased bone should not be extracted from the hip-joint, in cases where the secondary disorder is in an early stage.

Cases sometimes end fatally from this complication after long suppuration from excision of a joint, in which there can be little doubt that at the time of the operation indications of the change would have been found if sought, and in which, had amputation been then performed, there might have been room to hope that the mischief had not attained sufficient extent to prevent recovery, or interfere with subsequent health. Considering that the early stages of this disorder are often without obvious symptoms, it becomes necessary, in every case where conservative surgery is contemplated, to inquire somewhat closely into the condition of the patient. With regard to the urine, whether it has increased in quantity or become ever so slightly albuminous; whether there is thirst, any enlargement of the liver or spleen, any tendency to diarrhœa, or any trace of œdema; the finding of any of these indications of lardaceous disease—or even only if, after long and large suppuration, the aspect of the patient be especially pallid and worn—must suggest the wisdom of giving pause to the discharge in the manner which, regardless of other considerations, is the most speedy and the least exhausting.

The various forms of suppuration which belong to internal organs, and chiefly come under the physician, are less under control than those which are exposed to the more superficial art of surgery. When the disease comes on from phthisis the cause must be regarded as almost beyond obviation; but it is to be sometimes noticed that, with the establishment of the secondary disorder, the first tends to quiescence. When the disorder has sprung from dysentery, that disease, usually of tropical origin, may be a matter only of the past when the lardaceous condition attracts notice. And with regard to other sources of suppuration it too often happens that, either by time or place, they are put out of reach.

With a present drain which cannot be stopped, our endeavors must be directed to compensation; and I am assured that, whether in regard to diseases of bones and joints or internal suppurative lesions, much may be done in this direction by suitable diet and nutritious drugs. The mere chemical loss in suppuration can be restored by thus supplying the salts—chiefly those of potash—which pus removes; while, to make up for the organic or corpuscular loss by fresh supply, the treatment and surroundings of the patient should be adjusted to secure by every means general health and vigorous nutrition.

The diet should be generous and varied, and beef-tea and Liebig's extract of meat may be added, for the sake of their saline constituents. Under profuse suppuration alkalies are generally well borne, and I have satisfied myself that its effects may be mitigated by the use of potash and its salts, together with iron and quinine. I have come to the habit of using in such circumstances a mixture of liquor patassæ, phosphate of potash, and tartrated iron; together with cinchona and quinine, most conveniently given in an alternate mixture. Cod-liver oil may be superadded and suitably made a vehicle for the liquor potassæ. Alkalies should not be given so as to make the urine alkaline, nor pushed independently of the ordinary tonics and restoratives.

Coming now from prevention to the cure or mitigation of the established disease, the action of iodide of potassium upon the disorder, when of syphilitic origin, is perhaps the most striking fact in its therapeutics. An instance of rapid diminution of the swollen viscera, and corresponding improvement in general health under this remedy, is related at page 217. Considerable doses of the iodide of potassium should be given, generally suitably associated with iron in some shape, perhaps best as the iodide.

When the disease is of syphilitic origin much is sometimes to be done even in way of cure by such specific treatment; but it is to be recognized that the disorder is less amenable in its renal localization than as it affects organs other than the kidney.

Passing to the more frequent type of the disease in which it has had its source in suppuration, the question which first presents itself is, whether the benefit found from the administration of potash is such as the pathological facts—notably the extraordinary solubility of the morbid deposit in alkalies—would lead us to anticipate. It might be hoped so to charge the blood with alkali as to make it a solvent for the deposit; while a plenitude of potash could not fail to prevent any change consequent upon its deficiency.

But disease cannot be treated in a test-tube. The deposit is not to be dissolved but by the alkalies in their caustic state; and though these in moderate proportion may be introduced into the stomach, it is by no means a matter of course that they will reach the tissues in the same shape. To appeal to experience—and I have given potash in this disorder largely and long, and in many forms and modes of admixture—I must say that the alkalies are well borne; that under their influence, together with that of iron, quinine, and time, patients will lose swelling, whether visceral or serous, gain flesh, and assume an improved aspect in all the phases of the disease; but in the advanced stages at least it is seldom susceptible of complete removal by any means at our command, the nearest approach being obtained by the iodides when it is of syphilitic origin. On the whole the use of alkalies unassociated with tonics is disappointing, though tonics together with alkalies give better results than tonics alone. Where the disease is chiefly renal and of long standing no curative attempts are successful; secondary fibrosis, indeed, in these circumstances is seldom absent, and probably marks a condition from which there is no return. Where the disorder is chiefly declared by enlargement of the liver and spleen the chances of treatment are better; with these organs the question appears to be one more of infiltration than growth.

Some such mixture may be given as has been referred to in relation to prevention, or liquor potassæ may be administered before meals, with iron and quinine at stated intervals. Ferrum redactum, iodide of iron and cod-liver oil appear to be often suitable, while the good to be got from the remedies which issue from the dispensary of nature—fresh air and salt breezes—is such as to join witness with more artificial agencies in declaring the amenability of at least some of the forms of the disease.

Supposing we have to deal with the disorder chiefly in its renal manifestation, where the originating condition has become only a matter of history and the organic state is in its nature irretrievable, we may confine our attention to the mitigation of symptoms. Albuminuria of this type differs from that of other sorts in its lesser tendency to uræmia, its infrequent association with abnormal vascular tension or cardiac hypertrophy, and its greater proneness to wasting discharges and death by exhaustion rather than by poisons. Whatever, therefore, be the complication to be met, these considerations must give bias to the treatment. The diet may be more liberal then where uræmia is to be more feared, and iron may be used freely and constantly, since poverty of blood has to be overcome rather than the results of arterial fulness apprehended. Œdema, so constant in this disease, must be met by diuretics combined with ferruginous medicines—acetate of potash with acetate of iron, the bitartrate of potash with ferrum tartaratum, or digitalis with the perchloride. The dropsy of renal disease, whatever be its nature, is more constantly associated with anæmia than with any other condition, and perhaps there is no remedy more generally useful in the treatment of this symptom than perchloride of iron. With the qualifications which have been insisted on, the several forms of serous effusion which are apt to occur in this disease are to be treated on the principles which have already been laid down in reference to renal dropsy in general; the hydragogue purgatives may be used with advantage when accumulation in the serous cavities is a source of danger.

When, owing to the occurrence of diarrhœa, or to the abundance of the urine, no dropsical effusion exists, perhaps nothing more will be called for than such ferruginous or specific medicines as have been found to suit the individual case. The syrup of the iodide of iron, or the tincture of the perchloride, may be associated, should there be a syphilitic history, with iodide of potassium.

Where there is tubercular disease cod-liver oil will, of course, be resorted to.

Vapor baths are not so useful in this disease as with granular degeneration; the greater debility of the patient makes him less able to withstand their depressing influence, and the small tendency to uræmia renders them generally unnecessary.

But when uræmic symptoms threaten, as must needs happen now and then—for the purely lardaceous condition of early disease becomes complicated in time with tubal and intertubal mischief—they must be met with baths of hot air or hot water, elaterium, and the like, as if they had arisen in connection with renal incapacity of any other origin.

One of the worst complications of the disorder is the diarrhœa, and one remedy after another will often be tried with only temporary benefit. Opium may be used without fear. The greatest benefit will often result from giving ten or fifteen drops of laudanum with the ferruginous and diuretic medicines. The intolerance of opium, which is so strongly marked with granular degeneration, does not exist with this disease, or at least not to a sufficient extent to be a source of danger. If small doses of opium alone fail to check the purging they may be combined with astringents, as in the form of compound kino powder, or mixed with catechu, logwood, or the red gum of Australia. When such remedies fail we must have recourse to acetate of lead, and lastly to sulphate of copper, the latter being the most efficacious of all the drugs directed to this end. Both may be given with small quantities of opium.

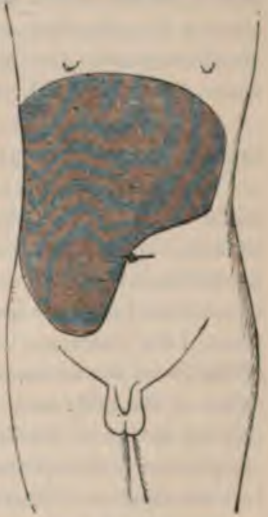
It is not necessary to follow in detail the treatment of the several inflammatory and other complications of the disease, since what has been said with regard to other renal affections applies also to this.

The following cases illustrate the pathology of the disease, as well as the line of treatment which it was thought advisable to adopt:

*Exposure to a Tropical Climate, Syphilis; and, as Subsequently Ascertained, Dysentery—General Lardaceous Disease, with Enormous Visceral Enlargement, chiefly of the Liver, together with Albuminuria, Diarrhœa, and Vomiting—Improvement under Treatment, with Diminution of Visceral Swelling and Recovery of Apparent Health—Death from Tubercular Disease Three Years later—*Post-mortem *Examination of the Viscera, with Evidence, in the Case of the Liver, Spleen, and Mucous Membranes, of their nearly Complete Restoration.*

George Hall, a retired soldier, thirty-five years of age, became my patient at St. George's Hospital, on October 17, 1866.

He had entered the army in the year 1852, served through the whole Crimean war, been sent to Madras in 1857, and invalided home in 1860. He said that while in India he never had had dysentery or even severe diarrhœa, but had suffered long from a bubo of syphilitic origin, which discharged persistently, but not profusely, for eighty-seven days. He subsequently had tender lumps on the ulnæ, near the elbows and on the shins, with pain, which increased at night. For the last two or three months he had vomited frequently, especially after food, and had lost much flesh.



On admission he had a sallow skin and sharpened features, attenuated limbs, and a large belly. The abdominal swelling depended upon a great increase in the size of the liver, the outline of which could be felt with perfect distinctness through the thin and stretched integuments. This organ occupied the greater part of the abdominal cavity, reaching on the right side nearly to the groin, while on the left it extended far into the left hypochondrium, where the tumor occupied the position of the spleen, and may have been continued in this situation by that organ in a swollen condition inseparably applied to the hepatic mass. Whether wholly hepatic or partly splenic, no notch or furrow could be felt to break the continuity of the enlargement. On the right side the mass reached vertically downward for fifteen inches below the nipple; on the left for three inches below the margin of the ribs. Its outline, as ascertained by percussion and touch, is represented.

There was no evidence of fluid in the abdomen, though the ankles were slightly œdematous. There was some thirst, and the tongue was red and dry. The urine was abundantly albuminous, of rather high color, though of low specific gravity—1009. It was acid, contained large strongly marked hyaline casts, and for the twenty-four hours ending on October 18th, gave the following quantitative results:

Quantity = 1530 c.c.
Urea = 18.36 "
Uric acid = .612 "
Phosphoric acid = .382 "
Sulphuric acid = .828 "
Chlorine = 2.280 "

The heart was lifted in position by the abdominal tumor; it was evidently small in size, nor was the pulse otherwise than soft.

Treatment was mainly directed by the syphilitic taint, which was equally evident in history and symptoms; 10 grains of iodide of potassium with a drachm and a half of the syrup of iodide of iron were given three times a day, together with a scruple of acetate of potash, partly as a means of administering this alkali, and in part for the sake of the diuretic action which was indicated by the œdema. The patient had good diet, with wine.

Beyond the stated remedies he required small opiates from time to time, to check a somewhat obstinate watery diarrhœa.

Within three weeks of the commencement of this treatment the abdominal swelling had palpably lessened; the lower margin of the enlarged liver had been marked upon the skin with nitrate of silver, so that the lifting of the lower edge could be strictly appreciated, and by December 5th, it was found that the margin of the tumefaction both on the right and left sides of the abdomen (belonging, as was thought, to the right and left lobes of the liver) was two inches higher than on admission. With this the prominence of the belly had greatly lessened, evidently to the eye and measurably by means of his recovered power of buttoning his garments. The complexion had improved, he had gained flesh and strength, the appetite had returned, and diarrhœa and vomiting, with both of which he had been much troubled, had nearly ceased. He thought himself able to resume his duties, as attendant in an asylum, and was accordingly made an out-patient, the same treatment being continued.

While in the hospital his temperature had been habitually low, ranging from 97.1 to 97.4.

He came to show himself from time to time, with constant and rapid improvement, and at last ceased to attend, thinking it no longer necessary to do so.

On June 2, 1869, I recognized my old patient again in the hospital under one of my colleagues. He had just been re-admitted and was evidently moribund; his features were sharp and his frame attenuated, and it was not possible to examine him further than to ascertain that the belly instead of being swollen was retracted, while the liver now extended only two inches below the margin of the thorax. Next day he died.

When the body was examined a considerable amount of quite recent tubercle was found at both apices, surrounded by hepatized lung. In the right was a small recent vomica. The heart was natural in size and structure.

The transverse colon was much thickened, and presented on its mucous surface dense cicatrices which intersected each other in every direction, giving a honeycombed appearance like that of the mucous membrane of the stomach. There was here no ulceration; but on the ascending colon, where were no cicatrices, were many small clean-cut ulcers. The small intestine was natural in all respects—it gave no lardaceous reaction.

With regard to the organs which had given evidence during life of lardaceous change the liver was still larger than natural, weighing 6 lb. 2 oz. It projected two inches below the ribs. It was of natural consistence, and save that it was speckled with fatty degeneration was of natural color. It had not the elasticity or the translucency of lardaceous disease, and on the application of iodine gave no very evident sign of this change. Close, and more especially microscopic inspection, showed here and there small dots of distinctly lardaceous reaction; these, however, would have escaped notice had they not been somewhat painfully sought. The microscope showed a great excess of oil, and an increase of the hepatic fibrous tissue was apparent both to minute and rough observation.

The spleen weighed 15 oz.; it was intersected with thickened fibrous septa, the intervening tissue being firm, uniform, and waxy in appearance, closely resembling hard damson-cheese. It gave, however, no reaction with iodine.

The kidneys weighed together 13 oz. They were smooth externally, save that their natural gloss was replaced by a sandiness of surface. The capsules were slightly adherent. The cortices were in about their normal proportion to the cones, white and uniform. The malpighian bodies glistened and gave the iodine reaction in a very characteristic manner, as also did the straight vessels of the cones. A few small cysts were found in both cones and cortex.

No lardaceous reaction was found upon the mucous membrane or anywhere save in the kidneys, and to the small extent mentioned with reference to the liver.

The disorder in this case had a complicated origin, presenting among its antecedents more than one of the circumstances by which it is apt to be preceded. The patient had lived within the tropics; he had suffered severely from dysentery, as declared by the state of the bowels after death, though the occurrence of the attack had been forgotten, or had been obscured by some other illness; and he had had syphilis. The last was probably the leading cause; but it is not to be doubted that the dysenteric discharge must have largely contributed; while the influence of both was brought to bear with the maximum of effect under the condition of malnutrition which an equatorial climate engenders in an Englishman.

The great interest of this case, however, is in regard to the amenability to treatment or tendency to recovery which the lardaceous disorder presents. The visceral enlargement had been extreme, the right lobe of the liver covering the whole of the right side of the abdomen. The thinness of the integuments allowed the outline of the swelling to be ascertained with more than common distinctness, and the diminution to be traced from week to week. While the man was under observation the lower edge lifted at the rate of rather more than an inch a month; and the tangible size of the liver at last showed that within the space of three years it had gathered itself up from the line of the groin to within two inches of the thorax. How many pounds of lardaceous matter were removed in this process it is idle to speculate—necessarily a considerable number. The shrinking, so far as we can judge by the state of the liver after death, was due rather to a process of true recovery, removal of the infiltrating matter, than to the cirrhotic contraction which lardaceous organs are apt slowly to undergo. The recovery was to all appearance mainly due to iodide of potassium, the use of which was sufficiently hinted by the occurrence of syphilis among the complicated antecedents of the attack.

It is worth noting that, as is generally the case, the lardaceous affection of the kidney proved more persistent or less amenable than that of the other organs. It will be observed with regard to the bowels that though the obstinacy and character of the diarrhœa appeared to show that at one time these were involved, yet at death they had so far recovered as to give no reaction.

The tubercular disease, which at last proved fatal, was apparently of subsequent origin to the lardaceous, and had no obvious connection with it.

*Disease of Pelvis, with Suppuration—Consequent Lardaceous Enlargement of Liver and Spleen, with Affection of Kidney and Bowels—Œdema, Diarrhœa, Epistaxis—Analysis of Urine—Temporary Improvement under Treatment—Death—*Post-mortem *Examination.*

Isaac Gilbert, thirteen years of age, was frequently a patient in St. George's Hospital, successively under Mr. Pollock, Dr. Barclay, and myself.

He came under medical observation October 17, 1866. He had long been under surgical care for disease of the pelvis. He had an open sore, which for the last four years had discharged pus freely from the neighborhood of the sacrum, while another opening was in connection with each ilium. For a year there had been œdema of the legs; swelling of the body for somewhat longer.

The boy was very pallid. The swelling of the belly was evidently due to an increase in the size of the liver, which could be clearly felt as low as the umbilicus. The feet, legs, and genital organs were œdematous. There was cough, slight mucous expectoration, exaggerated breathing, and some want of resonance at the left apex. The bowels were loose, the tongue rather dry, and there was complaint of thirst. The nose frequently bled.

The urine in twenty-four hours amounted to 22 oz., or 680 c.c. It was acid. The albuminous clot occupied half the bulk of the fluid. Delicate transparent fibrinous casts were found, some of which contained epithelial cells, others oil-globules.

The following are the amounts of the several constituents passed during twenty-four hours:

Urea	9.36	grammes.
Uric acid	.149	"
Phosphoric acid	.136	"
Sulphuric acid	.51	"
Chlorine	1.08	"
Soluble ash	2.516	"
Insoluble ash	.163	"
Potash	.707	"
Soda	.802	"

The patient shortly after this date became an out-patient, and took persistently cod-liver oil with a mixture containing liquor potassæ and liquor sodæ, besides astringents or diuretics, when called for by the diarrhœa or œdema.

He seemed to improve for a time, but in February the dropsy again increased, and he was re-admitted. The belly was now tense and prominent, the surface covered with large veins. Hard masses could be felt in the position of the liver and spleen. The œdema was considerable over the lower extremities, scrotum, and penis. The bowels were loose. The discharge continued, and he was in much the same state as when first seen. The urine was highly albuminous (alb.$=\frac{1}{2}$). It is not necessary to follow the treatment in detail. His diet was of a liberal kind, including eggs and gin. He took salts of iron, cod-liver oil, and the alkalies. Liquor potassæ was given in solution, associated with tartrate of iron; or, when a diuretic action was needed, acetate of potash and acetate of iron were given together, while the alkalies were combined with the cod-liver oil. Three drachms of cod-liver oil, with liq. potassæ, ♏ xv., the same quantity of liq. sodæ, and a sufficiency of water, formed a mixture which was taken for a considerable time without objection. The diarrhœa was held in abeyance by the addition of a few minims of laudanum to the ferruginous medicine. Under these measures the health improved in every respect, the œdema almost disappeared, and the pallor diminished. He, however, got tired of the hospital, and left on April 5. On May 7, as I was informed, he sank somewhat suddenly.

It was not possible to make a complete examination of the body; but under circumstances of difficulty an incision was made in the abdomen, and a portion of liver and small intestine extracted. The peritoneum was occupied by recent adhesions, peritonitis having probably been the cause of death. The liver and spleen were both greatly enlarged. A portion of the former which was taken out was highly waxy in appearance, and with iodine gave the test-color in the most intense manner. The affected tissue came out as black spots, separated by a network which gave no reaction. The mucous membrane of the coil of intestine removed was affected in an equally characteristic way.

The case calls for few remarks. It is almost typical. The improvement under treatment was very decided, notwithstanding that the extensive disease both of the pelvis and of the viscera gave a very unpromising character to the case. The diarrhœa was completely under control, although, as ultimately proved, the change in the intestine was extreme. There were no uræmic symptoms from first to last. Death was apparently due to peritonitis, a complication which, though less common than pneumonia, is fatal in a considerable portion of cases. Epistaxis, though not uncommon in this disease, is less frequent than with the granular kidney.

CHAPTER XIV.

ON THE CONDITION OF THE HEART AND ARTERIES IN CHRONIC RENAL DISEASE.

Bright noticed hypertrophy of the left ventricle as associated with disease of the kidneys, and attributed it to a difficulty with which the blood traversed the general capillaries by reason of the impurity due to defective renal action. But this simple view has not constantly held its ground. By later observers the cardiac hypertrophy was somewhat unintelligibly explained, not by any change in the quality of the blood, but merely by the resistance which it encountered in passing through the diseased kidney. The renal arteries, it was urged, were short, wide, and but at a short distance from the aortic orifice, and must admit so large a share of the blood of the descending aorta that any impediment in their course must quickly react backward upon the heart and excite it to increased effort and consequent hypertrophy. This view, however, appears to have been tacitly abandoned; and, indeed, the obvious improbability that even the total occlusion of two comparatively remote and small arteries, which together can but transmit a very small fraction of the blood which leaves the ventricle, should produce sufficient repletion of the arterial system to tell as far back as the heart, is such that it probably now has no supporters. The iliac artery may be tied without any such result. Were the ventricle hypertrophied to suit the altered state of the merely renal circulation, its thickness would be disproportioned to the state of the circulation in every other organ. Dismissing this view, therefore, we come to later days and still conflicting opinions.

In addition to noting the hypertrophy of the left ventricle, and inferring its cause in capillary retardation, Bright had observed that the renal arteries, as seen after death, and the radial, as felt during life, were often the seat of morbid change. Dr. George Johnson added importantly to these observations, and in a paper read before the Medico-Chirurgical Society in the year 1867 pointed out that an arterial thickening was general to the whole body in certain cases of chronic albuminuria, and expressed views as to its nature and mode of origin which have since become the subjects of discussion. His views are briefly these:

The small arteries in chronic Bright's Disease, generally throughout the body, undertake a hypertrophy of their muscular coat as the result of a continued overaction on their part, whereby they, acting as stopcocks, to use Dr. Johnson's phraseology, endeavor to exclude the contaminated blood from the tissues. The heart becomes hypertrophied by its efforts to overcome the resistance thus occasioned. The heart and the arteries thus exert themselves in antagonism to their mutual hypertrophy.

Not as yet to discuss these views, I will proceed to the next phase in which the subject has been presented.

Sir W. Gull and Dr. Sutton, in a paper read before the same Society in the year 1872, point out what Dr. Johnson, with a rather surprising adhesion to his earlier views, had in his communication denied, namely, that the granular kidney has its origin in intertubular fibroid growth. They show that the thickened arteries are usually associated with this fibrotic renal change, and maintain that the thickened arteries do not owe their increase of parietal bulk to true hypertrophy of muscle, but merely share in the process of fibroid overgrowth which in most of the instances adduced is the essential renal change.

Thus in their view the process by which the vessels are thickened is not a hypertrophy of arterial muscle secondary to and consequent upon renal change, but part of a condition of general fibrosis, which affects simultaneously many organs and tissues, belongs especially to the vascular system, and tells upon that part of the renal structure which is in closest relation to the arterial channels only as part of a general scheme of vascular deterioration.

They represent the affected arteries as thickened rather in their fibroid than their muscular coats, while the latter are virtually atrophied and degenerate.

To state their conclusions in their own words they are as follows:

"1. There is a diseased state characterized by hyalin-fibroid formation in the arterioles and capillaries.

"2. This morbid change is attended with atrophy of the adjacent tissues.

"3. It is probable that this morbid change commonly begins in the kidney, but there is evidence of its beginning primarily in other organs.

"4. The contraction and atrophy of the kidney are but part and parcel of the general morbid change.

"5. The kidneys may be but little, if at all, affected, whilst the morbid change is far advanced in other organs.

"6. This morbid change in the arterioles and capillaries is the primary and essential condition of the morbid state called chronic Bright's disease with contracted kidney.

"7. The clinical history varies according to the organs primarily and chiefly affected.

"8. In the present state of our knowledge we cannot refer the vascular changes to an antecedent change in the blood due to defective renal excretion.

"9. The kidneys may undergo extreme degenerative changes without being attended by the cardio-vascular and other lesions characteristic of the condition known as chronic Bright's disease.

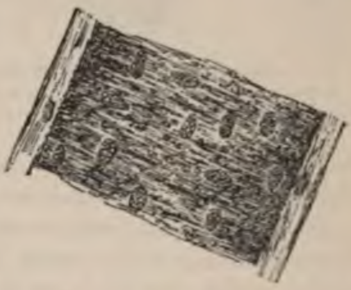
"10. The morbid state under discussion is allied with the conditions of old age, and its area may be said hypothetically to correspond to the 'area vasculosa.'

"11. The changes, though allied with senile alterations, are probably due to distinct causes not yet ascertained.

"Should it be considered necessary," say these authors in conclusion, "to distinguish this morbid state by any special term, we venture to suggest for that purpose the name *arterio-capillary fibrosis*."

Since these questions have arisen some time has elapsed, and many opportunities for observation have presented themselves. With the advantage of such opportunities, and profiting at the same time by the labors of others where my own knowledge is most defective, I will endeavor to present the facts of the case with as much truth to nature as my means of observation permit. The questions involved may be conveniently arranged under three heads: firstly, the anatomical nature of the arterial change; secondly, the circumstances in which it occurs; thirdly, the nature of the process, as far as can be fairly conjectured, by which the alteration is brought about.

First, as to the anatomical nature of the arterial change.

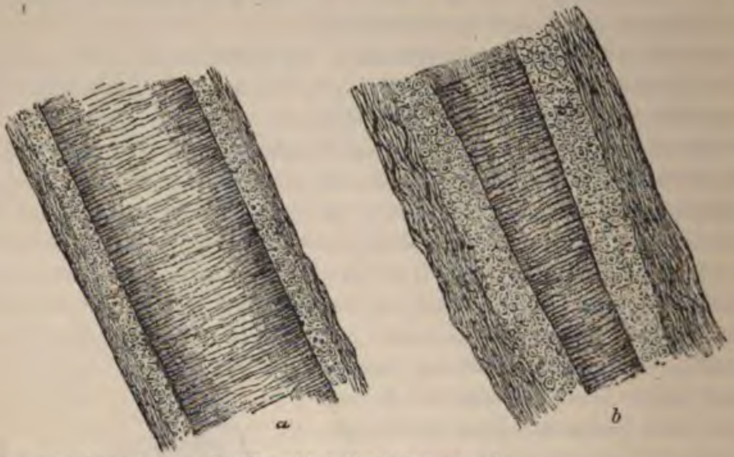


From pia mater, case of diffuse nephritis of ten weeks' standing. (See case of R. Warren, page 45.)

The affected vessels, which are apt to be the smaller arterioles, are often thickened in a sufficiently striking manner; this thickening involves both the muscular and fibrous coats.

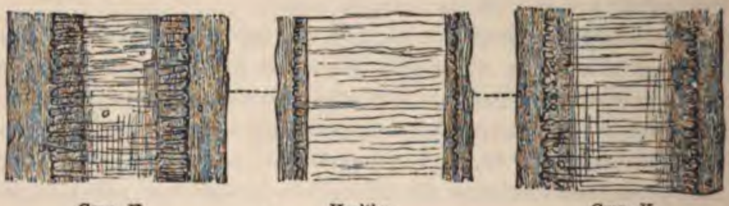
That the muscular coat is increased in bulk I think admits of no doubt; nor is it less evident that with its increase of bulk it undergoes change of texture. As the coat thickens the nuclei shrink, lessen in number, and finally disappear, being replaced or obscured by coarse transverse fibrillation. The coat thus altered refuses carmine in a manner which at once distinguishes it. With the first recognizable change are frequent evidences of fatty degeneration. Added to these, but as a later sign of altered structure, the affected muscular coat often acquires a lining, which, under the microscope, is conspicuous as marking the channel, consisting of fatty, more or less intermixed with crystalline elements.

Next as to the outer fibroid coat or tunica adventitia. The thickening of the arterial sheath, which was pointed out by Sir. W. Gull and Dr. Sutton, and to which they attach so much importance, does not seem to admit of dispute. Dr. Johnson considers the appearance to be a delusive result of reagents, but it is evident by whatever process the vessels are preserved, and even when they are looked at fresh from the body without preparation of any kind. In some of the specimens I have figured I have used pure glycerine, in others Canada balsam; but the question of reagents may be fairly put aside, since in the observations of Sir W. Gull and Dr. Sutton, as well as in those I have myself made, the diseased vessels have always been compared with healthy ones treated in precisely the same manner. The fibroid sheath becomes wavy and thick, and it finally assumes an appearance as if swollen with translucent structureless exudation, which transforms its normally thin vesture into a bulky outer cylinder which may equal or even surpass the thickness of the inner tube.



a A corresponding artery from healthy subject, identically prepared.
b Free artery from subarachnoid space, from brain of Tillett (see page 140), showing the thickening of both coats. The specimen was hardened in chromic acid and preserved in Canada balsam.

As early evidence of the morbid state change in the texture of the muscular coat—loss of nuclei, coarse fibrillation, and spotting with oil—it is to be more surely appreciated than increase of its thickness or of the thickness of the fibroid sheath. The thickness of wall in relation to calibre may be partly regulated by the state of vascular contraction at death or produced



Gran. K. Healthy. Gran. K.
(In glycerine) 180 diameters.

afterward by rigor mortis; but it would seem that all variation in this respect is less than might be thought likely. The regularity with which the arterial repeats itself in conjunction with the renal state is such as to show that the parietal thickness generally depends upon constant, not accidental conditions.

The muscular coat appears usually, though not always, to increase before the fibrous, and in some cases its abnormal bulk is the most noticeable alteration. There are others—and these are cases of long standing (I have noticed it after death by apoplexy)—in which degeneration has proceeded to an extent to mask or supersede every other change.

The vascular change is associated so constantly with hypertrophy of the left ventricle that the latter, if independent of valvular disease or tangible obstruction, may in a case of renal disease be generally accepted as evidence of the former.

Taking this test, it appears that the change in question is, with rare exception, constantly present when the kidneys are granular; that it occurs with tubal nephritis when that disorder is at all chronic; and that it is generally absent when the kidneys are lardaceous.

(In glycerine) 375 diameters. Artery of pia mater after death by cerebral hemorrhage with granular kidneys.

I have elsewhere shown (page 130) that uncomplicated hypertrophy of the heart was found after death in 45 per cent. of cases of granular degeneration.

This statement, at first sight, gives the frequency of hypertrophy as much too small, as in many of the cases on which it is based there existed valvular disease or pericarditis, in addition to hypertrophy, which cases are, of course, not enumerated under the category of uncomplicated hypertrophy. Excluding them altogether from consideration, the absolute number would stand thus: Of 42 instances of granular degeneration without valvular disease or pericarditis there existed marked hypertrophy of the heart in 31; or in a proportion of 74 per cent.

Of tubal or diffuse nephritis, hypertrophy, as will presently appear, is also to be recognized as a general accompaniment, though commonly present in a lesser degree than when the kidneys are granular, as having had less time to develop. The older tables fail to do justice to this concurrence.

Of the cases of unmixed lardaceous disease simple hypertrophy existed but in 2 per cent.

To compare the experience of Guy's Hospital with that of St. George's, Dr. Galabin[1] found that of 66 cases of granular degeneration in which there was no other reason to account for cardiac hypertrophy this existed in 53 instances. In 13 cases of pure lardaceous disease the heart was hypertrophied but in 1. With tubal nephritis Dr. Galabin states that of 22 cases simple hypertrophy existed in 11, or exactly half the number. It is probable, however, that in many or most of these cases the tubal had become complicated by intertubal change.

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[1] Dr. Galabin made the condition of the heart and arteries in Bright's disease the subject of an inaugural thesis at Cambridge, which I had the advantage of listening to, and which has since been published. The subject is there treated with much ability, and I find myself very much in accord with his conclusions.

Hypertrophy of the heart may generally be assumed, and I have so taken it, as an index of the cardio-vascular change. Arterial thickening, indeed, is more constant after death with renal granulation than is the cardiac. This condition of vessel, as found in the pia mater, is so continually found with this state of kidney that the exceptions have all the interest of curiosities. Putting aside the cases where the thickening had given place to degeneration, I can recall but one instance in my own knowledge where these vessels were healthy while the kidneys displayed advanced disease of the kind in question. A young woman died at the age of 22 in a state of coma—uræmic, as it turned out—which had come on with little premonition. She was not known to have had dropsy or any of the common symptoms of renal disease, though before her death it was ascertained that the urine was albuminous.

The kidneys were in an extreme state of fibroid contraction, with roughened but not regularly granular surfaces; they together weighed but 3 ounces 3½ drachms. The heart was decidedly hypertrophied, the left ventricle relatively thick, while the weight of the organ was 12 ounces, a large weight for the body, which was unusually small. The arteries of the pia mater were absolutely natural; the nuclei large, neither muscular nor fibroid coat more than normally thick, while the nuclei, both transverse and longitudinal, were bulky, plentiful, and distinct.

This case was strikingly contrasted with that of another young woman, also 22 years of age, who died at nearly the same time with granular and fibrotic kidneys, which weighed together only 3¾ ounces, almost exactly the same weight as in the first instance. The heart in this case weighed but 8¼ ounces, so that it was not appreciably hypertrophied, but the vessels of the pia mater were markedly so; the muscular coat thick and coarsely fibrous in appearance, to the obliteration of the nuclei; the proper fibrous coat also exaggerated.

This pair of cases may serve to show that one part of the circulating system may be affected by the hypertrophic process and not another. In both there was abundant cause for uræmia, and presumably for hypertrophy of heart and vessel; but in the first the heart was affected, and not the arteries, or at least not those of the pia mater; in the second the arteries, and not the heart.

Drawing no inferences as yet from these statements beyond pointing to two facts which are not to be questioned, namely, that hypertrophy of the left ventricle is rarely absent when the kidneys are granular, rarely present when they are lardaceous—I will add some details which may throw light on the nature of the connection between the cardio-vascular and the renal change.

The first point to determine may be simply put—is the vascular change directly produced by the renal disease with which it is associated, or is it part and parcel of a general fibrosis in which the kidneys participate, but as common sharers in a general process of fibroid overgrowth? Is the renal change the cause of the vascular, or are the two but common results of a widely acting morbid agency?

To see the facts in their barest form, the experiments of nature in their greatest simplicity, we must appeal to the pathology of childhood; and here, being able to put out of the question not only actually senile changes, but as a rule the degenerations, whether fatty or fibroid, which are common to many organs and tissues, and by which old age is as it were anticipated, we may trace the results of the disease we are studying without any admixture with changes which do not properly belong to it and are associated with it only accidentally.

Looking at the matter from this point of view, I will epitomize the results I have obtained.

Children sometimes, though not often, suffer from granular degeneration of the kidney. With them the disease takes its most typical shape: the new fibroid growth in the kidney is abundant and highly nucleated, the destruction of tubes great, and the symptoms correspondingly marked. In these cases the vascular change is extreme and characteristic, the hypertrophy of the left ventricle great, and cerebral hemorrhage an occasional termination.

Emily Tillett, whose case has been related at page 140, died at the age of twelve years with granular kidneys and cerebral apoplexy. The heart weighed no less than 8½ ounces, its increase of bulk being due to simple hypertrophy of the left ventricle. The arteries of the pia mater and of the kidney were greatly thickened both in their muscular and fibroid coats, dotted with oil, and irregular in contour. An artery from the subarachnoid space, which shows the thickening both of the muscular and fibrous coats, is represented at page 226.

Charles Dodd, whose case has been given at page 143, died at the age of fourteen, of cerebral uræmia consequent upon granular degeneration of the kidney. The heart weighed 8 ounces, the increase of bulk being due to simple hypertrophy of the left ventricle. The arteries of the pia mater were greatly thickened, both in their muscular and their fibroid coats, and were in an advanced stage of fatty degeneration.

William Smith died at the age of eleven, under the care of my colleague, Dr. Ogle, at St. George's Hospital. He had had good health until three years before his death, when he had scarlatina severely, and was never well afterward. He remained unable to work, and was constantly subject to headache, vomiting, and drowsiness. Latterly he lost his appetite and became thin, and for about two months before his death was observed to pass a large quantity of pale urine. Subsequently his face became œdematous, and he suddenly lost his sight; upon which he was sent to the hospital. He was pale and wasted, his face only puffy. His gums and mouth were ulcerated, and a diphtheritic membrane was visible in the fauces. He had headache, vomiting, and diarrhœa. He was totally blind, save that with the left eye he could recognize the position of a strong light. The pupils were dilated. Mr. Carter reported, as the result of ophthalmoscopic examination, that both eyes were affected by albuminous retinitis, and that the retinæ had become very extensively detached, the detachment explaining the sudden access of total blindness. The urine was pale, albuminous to one-half, had a specific gravity of 1008, and contained granular and hyaline casts. He had several attacks of bleeding at the nose; and the increased dulness and the distance of the cardiac sounds gave evidence of much hypertrophy of the heart. He sank after he had been in the hospital three weeks, the loss of blood by the nose, which eventually had to be plugged, contributing much to the fatal issue.

The kidneys were granular on their surfaces, and were dotted with spots of extravasation; the cortices were shrunk; and the appearances characteristic of granular degeneration. The pair weighed 5 ounces. Dr. Ogle secured me an opportunity of examining the organs microscopically, and I found them to present exquisite specimens of the comparatively rapid form of the disease which appears in childhood, with a profuse growth of nuclear tissue in the intertubular or perivascular districts of the organ. The tubes were affected by pressure; some compressed, others irregularly dilated. The renal arteries were enormously thickened, and their coats in some instances fatty. The heart weighed 8 ounces, its increase of weight being due to hypertrophy of the left ventricle. There was no valvular disease. The brain was anæmic and watery, with about half an ounce of clear fluid within each lateral ventricle. The retinæ were covered with characteristic spots of opacity and extravasation; they were separable from the choroid with unnatural facility.

The pia mater was not examined, but this was not necessary to establish the existence, in an extreme form, of the cardio-vascular changes which belong at every time of life to the granular kidney. The hypertrophy of the left ventricle, the thickening of the renal arteries, and the evidences of arterial degeneration in the epistaxis, and the condition of the retinæ, are together conclusive.

Through the kindness of Dr. Barlow,[1] of Manchester, I had an opportunity of examining a granular contracted kidney from the body of a little girl who had died, after repeated uræmic convulsions, at the age of five years and eleven months—the earliest instance of the disease with which I am acquainted. The kidneys were extreme instances of destruction by means of intertubular fibroid growth, which was abundant, highly nucleated, and collected in bulk under the capsule and around the arteries. The arteries of the kidney were themselves enormously thickened in their proper coats, and were surrounded by the newly-formed nucleated fibroid tissue. The heart was not weighed, but the left ventricle was stated as much enlarged and pale, the walls being relatively thin. The valves were healthy. There was no opportunity of examining the vessels of the pia mater. The characteristic changes of the kidney and its arteries, together with the enlargement of the left ventricle, were such as to show without much room for doubt that even at the exceptionally early age at which the disease proved fatal the blood-vessels were affected as they are when the disorder occurs later in life.

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[1] This case has been fully related by Dr. Barlow in the Lancet for August 1, 1874.

These instances—and with them I may allude to one of the same kind in which death occurred at the age of nine years, which is mentioned by Sir W. Gull and Dr. Sutton in their paper in the "Medico-Chirurgical Transactions," prove that with the renal fibrosis, let it occur as early as it will, we have necessarily—or at least invariably, within our experience—the characteristic vascular changes.

And it is to be observed that these vascular changes accompany the granular kidney not only when this disorder has come on spontaneously or from some constitutional influence, but when, as in the case of Smith, it has succeeded upon scarlatina. And it is equally to be observed when the disorder has arisen from an influence still more distinctly local. There is reason to believe that granular change in the kidney is sometimes sequent upon long-continued distention of the pelvis from stone. There was a suspicion of this sort in the case of Dodd, lately referred to; and I have seen another instance in a youth of 19, where marked granular degeneration with a formation of intertubular nuclear tissue and the characteristic changes in the circulating system were associated with great dilatation of both pelves, obviously of calculous origin.

Such cases connect the whole pathological series with a local almost to the exclusion of any general or constitutional origin.

We may now proceed to another and different class of cases—that in which the kidneys, previously healthy and belonging to young and previously healthy persons, have become the subjects of more or less definite acute inflammation. In these instances the disorder has usually been traced to scarlatina, cold, or one of the recognized causes of renal inflammation, and has run the course of acute dropsy. It is not possible absolutely to dissociate these cases from renal fibrosis, since with suitable methods more or less multiplication of the interstitial nuclei can almost always be detected; but this hypernucleation is clearly but a part, whether originally or by acquirement, of the local inflammation, and cannot by any stretch of imagination be looked upon as belonging to a common or widely spread fibrosis.

Thus, in the examples about to be related, we have to deal with disease special to the kidney, and only with such arterial and cardiac changes as directly result from it.

Beginning with the more chronic:

Catherine Burr, aged nineteen, came under my care, in St. George's Hospital, with general œdema, highly albuminous, blood-tinged, and scanty urine, and the general symptoms of acute renal dropsy in a severe shape. She had been suddenly attacked five weeks before with headache, vomiting, and febrile symptoms, and became dropsical a week later. She had not previously had scarlatina, and it was surmised that the attack was of this nature, though the evidence was by no means complete. Not to follow the details of her illness farther than to say that the dropsy became general and extreme, and then slowly lessened and disappeared, the patient in the later or non-dropsical stage became liable to severe headache, with partial loss of consciousness, but without convulsions. The radial artery was latterly distinctly tense, but the cardiac dulness not markedly increased. The first sound as heard over the septum was broken, almost reduplicated; the second loud and ringing. To these evidences of increased tension it is to be added that she had, as Mr. Carter was kind enough to ascertain, albuminuric patches and hemorrhages on both retinæ, which were productive of much dimness of sight.

Lastly a loud systolic murmur, with unusual action, became audible at the apex of the heart; the pulse rose to 160, the temperature to 102.4, and she quickly sank under a pyrexial attack of which the origin was not obvious. Death occurred ten months after the first symptom.

The kidneys weighed 19 ounces. The capsules were thin and easily detached, the surfaces smooth and ivory-colored, largely blotched with vascularity. The cortices were increased and yellowish; in the cones were some embolic wedges. Many of the tubes were irregularly distended, and contained thick plugs, others were perfectly natural. The interstitial tissue was uniformly and closely pervaded with large round nuclei. Much as the tubes had recovered themselves—marking their improvement probably by the subsidence of the dropsy—the intervening structure had participated too largely to allow of the recovery of the patient.

The immediate cause of death was purulent peritonitis, the abdominal cavity containing about a pint of brownish purulent fluid. The mitral valve presented at its base a ragged hole, which was in connection with a small pyæmic deposit which had formed in this situation. Much ragged fibrine had collected about the valve. The spleen contained an embolic block; and several hemorrhages the size of pins' heads, apparently of similar origin, were found in the left corpus striatum and other parts of the brain. Two or three small abscesses, evidently pyæmic, were found in the left lung. Thus, to dismiss what may be termed the accidental complications before proceeding to the necessary, suppurative peritonitis ensued—as it sometimes does, upon the renal mischief—upon this purulent absorption, and pyæmia, a pyæmic deposit upon the mitral valve, and the starting from thence of the embolic process.

The heart weighed 10 ounces; the left ventricle, besides the recent changes which have been adverted to, was slightly hypertrophied. The thickening of the arteries, as seen in the pia mater, was more marked. Both coats were considerably thickened, as shown in the woodcut, page 235, the outer coat conspicuously in vessels of about $\frac{1}{100}$ of an inch in diameter; the muscular, of which the nuclei were obscured, most so in those of smaller size.

Ethel Algar died under my care at the Hospital for Sick Children, at the age of six years. Two years previously she had had an attack of dropsy, which was preceded by catarrh in the head and chest, and was attributed to "catching cold." Six months afterward she again caught cold, with similar results, both as to the catarrh and the general œdema. Five months before her death the same series of symptoms resulted from a similar cause, and she came under notice with the characteristic symptoms of renal anasarca. The urine was scanty and highly albuminous, often discolored with blood, and contained fatty renal epithelium and large casts imbedding oil-globules. Without following the case in detail it may be enough to state that the dropsy almost disappeared under treatment, but never entirely, and that she eventually died after repeated uræmic convulsions.

The kidneys were typical examples of the mottled results of tubal nephritis. The pair weighed no less than 13 ounces. The surfaces were smooth, the capsule thin and unattached, the cortices enormously increased, and mottled with pink. There was no amyloid reaction.

The microscope showed the results of tubal nephritis as strikingly; the tubes, both straight and convoluted, were extensively plugged with large fibrinous cylinders, while in others the epithelium was irregularly accumulated. In addition to these changes there was a general overgrowth of profusely nucleated fibroid tissue nearly evenly throughout the whole organ. This was, perhaps, in greatest bulk around the malpighian bodies, but was generally of small mass, as an uniform and general overgrowth of the ubiquitous intertubular matrix. The arteries within the kidneys were much thickened, especially in their muscular coats. Those in other organs were not examined, but the left ventricle of the heart was much hypertrophied, the organ weighing 4½ ounces. There was no other change in the heart, save a few spots of atheroma on the mitral valve and aorta.

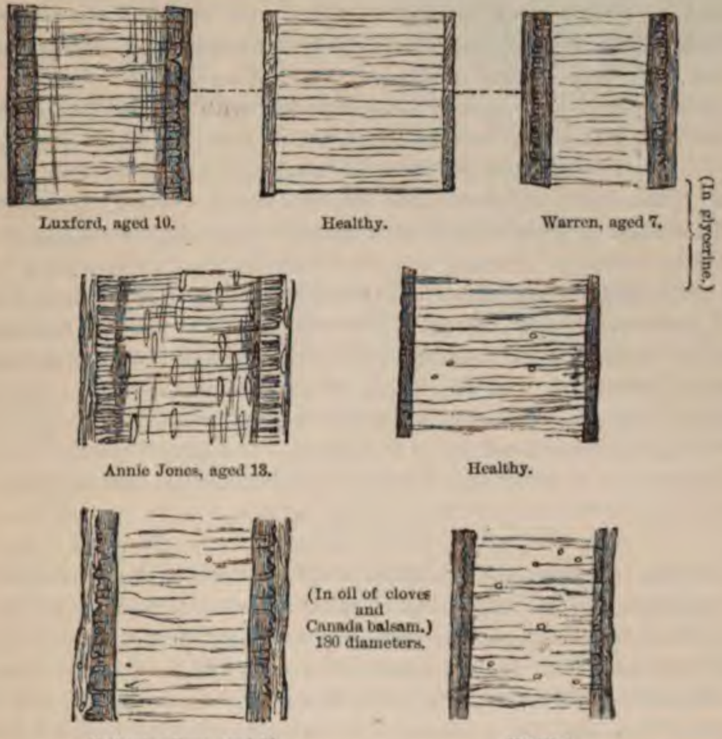
Annie Jones died under my care at St. George's Hospital, at the age of thirteen, with renal dropsy of four months' standing. The symptoms were those of acute tubal nephritis. The cause was not ascertained, but the outbreak of dropsy was sudden and general, the urine bloody and highly albuminous, and the casts numerous. The immediate cause of death was cerebral uræmia.

The kidneys, which weighed 12 ounces, were white, smooth, and mottled, and showed in a typical manner the results of tubal nephritis. The cortical tubes were irregularly filled with an excessive epithelial growth ; some contained blood ; and without more than ordinarily delicate methods no extra-tubal changes were to be seen. Logwood staining, however, displayed uniformly throughout the organ profuse nucleation of the interstitial tissue, with slight thickening. The heart weighed 9 ounces, the increase of weight being due to hypertrophy of the left ventricle. The valves were natural. The arteries of the pia mater were thickened, those most so which were of about $\frac{1}{300}$ inch in diameter ; the thickening affected chiefly the muscular coat, but the fibroid also to some extent. The muscular nuclei were obscured or wanting, but in other respects the evidences of degeneration were less marked than those of thickening. (See woodcut on opposite page.)

Harriet Luxford died at the age of ten years, of acute renal dropsy of little more than three months' duration. The kidneys, which were much enlarged, white, smooth, and mottled, displayed in translucent sections the most characteristic appearances of unmixed tubal nephritis ; the tubes were swollen by an extravagant and irregular epithelial growth, and many of them contained oil-globules ; beyond distention of the blood-vessels there was almost no change external to the tubes, but with logwood tinting and careful search several small spots of interstitial nucleation were discovered, enough to declare the tendency, though not as yet to interfere with the gland.

The heart weighed 6 ounces ; the left ventricle was hypertrophied, the valves healthy. The cerebral arteries were spotted with oil, the transverse striæ of the muscular coat were exaggerated, though the nuclei were generally to be seen ; and the muscular coat, particularly in arteries of about $\frac{1}{280}$ inch in diameter, was thickened—not greatly, but, as far as could be judged by comparison with healthy vessels, decidedly. (See woodcut on opposite page.)

Richard Warren, whose case is given in detail at page 45, died at the age of seven years, of acute renal dropsy, which had come on ten weeks previously. The kidneys were greatly enlarged, smooth, and mottled. In addition to the evidences of tubal disturbance there was slight general thickening, with superabundant nucleation of the intertubal tissue. The heart weighed 6¾ ounces ; it was free from valvular disease, but the left ventricle was remarkably hypertrophied; the musculi papillares were extraordinarily developed, and altogether the condition was more than ordinarily characteristic of renal hypertrophy. The arteries of the pia mater were in a marked state of fatty degeneration, which change had chiefly affected the nuclei of the muscular coat. Some thickening was detected in the muscular coat, and the fibrous sheath of many of the smaller arterioles appeared to be somewhat smooth and hyaline. (See woodcuts at page 226 and above.)



Arteries of pia mater from cases of diffuse nephritis, compared with healthy arteries identically treated.

John Mulcahy, aged seven years, came under my care at the Hospital for Sick Chidren, with severe scarlatinal dropsy; the œdema was general, the urine smoky and albuminous, and there was much dyspnœa, with evidence of congestion of both lungs. The pulmonary disturbance became more marked, and ultimately was the chief agent in causing his death, which took place nine weeks after the declaration of the rash, and apparently on the nineteenth day of the sequelæ, counting from a rigor which marked the beginning of the pulmonary disturbance, though it was not until three days later that œdema, first about the eyes, presented itself. The actual beginning of the renal inflammation cannot be fixed more nearly than between the appearance of the rash and of the dropsy ; at any rate less than nine weeks before death. Extensive hepatization of the right lung was found, and appeared to have been the chief cause of death. The kidneys together weighed 9 ounces ; with perfectly smooth surfaces, much injection showing through pale mottling, disproportionately swollen cortices and darkly congested cones, they were marked examples of recent scarlatinal nephritis. Microscopically they were equally characteristic, with swollen and irregularly abundant epithelium, distended tubes, and here and there signs, not too obvious, but evident on careful examination, of recent interstitial nucleation. This was not general, but here and there, where fibrous tissue normally existed in bulk, collections of circular apparently newly formed nuclei could be detected. Passing now to the circulating system, the left ventricle was strikingly hypertrophied ; the heart weighed $5\frac{3}{4}$ ounces ; the valves and great vessels were natural. The arteries of the pia mater examined in Canada balsam showed marked hyaline thickening of the outer coat, particularly in vessels of from $\frac{1}{700}$ to $\frac{1}{500}$ of an inch in diameter. The fibroid sheath of the affected vessels was thickened and its outline rendered irregular by apparently structureless exudation. The larger vessels were generally natural, nor could any muscular thickening be detected, save in the ventricle.

In these six cases the evidences of cardio-vascular change are sufficiently apparent. In five, slight but decided arterial thickening, for the most part involving the muscular coat, was found in the pia mater ; in one, in which the pia mater was not examined, a change of the same sort was found in the kidney. With regard to the heart this was enlarged to a marked degree in at least five instances. With a girl of 13 it weighed 9 ounces, about the average weight of the adult female heart ; with a girl of 10, whose heart should normally weigh about 4 ounces, it weighed 6 ; with one of 6, whose heart should weigh about 3 ounces, it weighed $4\frac{1}{2}$; with two boys, each 7 years old, and entitled to a heart of about 4 ounces, this organ weighed respectively $6\frac{3}{4}$ ounces and $5\frac{3}{4}$ ounces.

These cases, which represent a large class, appear to show conclusively that the cardio-vascular thickening, the nature of which has been discussed, is a direct consequence of simple renal inflammation. The inflammatory process, indeed, engenders in most cases where it proves fatal some degree —enough to be recognizable, if not to be injurious—of hypernucleation or incipient fibrosis, so that the dissociation of renal fibrosis and arterial thickening is not scholastically conclusive ; but the recent character of the fibrotic change, and its occurrence with abrupt symptoms in young and previously healthy subjects, place it practically beyond question that it belongs to the late and local disturbance, and not to any antecedent and general condition.

So far, then, *post-mortem* evidence appears to be conclusive as to the following points:

1. The cardio-vascular change is nearly constantly present, and usually in an extreme form, when the kidneys have become contracted and granular as the result of interstitial fibroid growth; this is the case, however young the subject, and although there may be reason to believe that the renal disorder has thus arisen at an early age as the result of scarlatina or stone.

2. Simple renal inflammation, essentially affecting the tubes, though productive of more or less interstitial nucleation, springing from a determinate cause and running a comparatively rapid course, is constantly succeeded by a lesser degree of the same cardio-vascular change, often more marked in the heart than in the arteries, but not admitting of question in either situation.

So far as simple nephritis is concerned, the direct production of the vascular change by the renal is made too obvious by the circumstances of the cases to need assertion. The youth and previous health of the subjects enable us to assume that but for the renal attack the heart and vessels would have been healthy; and it was further possible, in more than one instance, to trace the gradual enlargement of the heart under the influence of the renal disease. And, looking in this light at the class of precocious granular disease, it is not possible but to conclude that with this also the renal and the vascular changes are as simply cause and effect. The arterial and the cardiac thickening are the same in kind in both classes, however different in degree, and neither is the kidney of the one abruptly separable or distinguishable in nature from that of the other. The late nephritic and the early granular are the same. Tubal catarrh is associated with intertubal nucleation, nuclei develop into fibre, fibre contracts, and thus the difference between the nephritic and the granulating may be merely one of duration.

These facts point strongly, if not conclusively, to the view that the cardio-vascular change is not associated with the renal as a coeval fibrosis, but is directly produced by it. At the same time they indicate, not as an invariable law, but as a practical rule, to which there are few exceptions, this axiom—if the cardio-vascular change is marked, the kidneys have either primarily or secondarily undergone fibroid change; in other words, if the heart be hypertrophied the kidneys are the subjects of grave and probably intractable disease—a clinical landmark of no small value.

Light is thrown upon the association of renal and cardio-vascular change by cases in which one kidney has been destroyed or both damaged by disease not of the albuminuric kind.

Dr. Sibson has produced a numerical statement to the effect that in eight cases of renal calculus the heart was hypertrophied in five.

Dr. Galabin, in the thesis to which I have referred, relates two examples in which one kidney has been destroyed by calculous disease, and the heart in consequence became hypertrophied. To quote one: a man forty-three years of age died with cerebral hemorrhage. The right kidney was healthy, and weighed nine ounces. The left kidney was much wasted, and contained several calculi. The heart weighed twenty ounces ; the left ventricle was hypertrophied. The arteries of the brain were atheromatous, the aorta not so.

An apparently healthy young man was seized with acute pneumonia, of which he died, in St. George's Hospital. The cavities of both kidneys contained calculi, and were distended at the expense of the secreting substance, which in both was much atrophied. He was not known, however, to have had uræmic symptoms. The heart was natural in structure ; it weighed thirteen ounces ; the left ventricle was evidently thickened. The smallest arterioles of the *pia mater* presented hyaline increase of their outer coat, which in comparison with healthy vessels, similarly prepared (tinted with logwood and preserved in Canada balsam), did not admit of doubt. The larger vessels were perfectly natural, as in all instances were the arterial nuclei and muscle.

A man who lately died at the age of forty-seven, also under my care in St. George's Hospital, affords a further illustration of a similar chain of circumstances. He had of late suffered from frequent headaches, and had had several epileptiform seizures, but was able to do his work until the day of admission, when he lost the use of the limbs—first of the left, as was said—and quickly became unconscious and semi-comatose, without remaining evidence of lateral inequality. The urine could not be obtained, but there were signs of considerable hypertrophy of the heart, which, together with the character of the head-symptoms, were sufficiently suggestive as to the nature of the attack. He died in two days without recovering consciousness. The brain was simply watery—the attack obviously uræmic. Of the kidneys one only could be said to exist. The left had been destroyed, probably by calculous pyelitis, and nothing now remained of it but a multilocular shell with membranous walls, the cavity of which was filled with a soft material like putty. The right kidney was perfectly natural, save that it had undergone some degree of compensative hypertrophy, and now weighed eight ounces. The heart was natural, except some degree of disproportionate thickness of the left ventricle ; it weighed thirteen ounces. The vessels of the *pia mater* were examined under several modes of preparation. Those of smallest size were absolutely natural, the nuclei distinct, and the walls thin ; but above the diameter of about $\frac{1}{160}$ of an inch the increase of both muscular and fibrous coats was, though not extreme, quite decided on comparison with healthy vessels.

In this case it is to be observed that the compensative hypertrophy of the kidney was considerable, while the cardio-arterial changes, though declared, were proportionately small.

In these cases the neat hand of Nature has performed a conclusive experiment in answer to the question whether the changes which they display in the arterial segment of the circulatory system are part of a fibrosis common to vessels and organs, or are produced by glandular incapacity. In these subjects, conspicuously in the last, there was no suggestion of fibrosis in the state of any of the viscera, while the essence of the disease and the cause of death was simple abolition of renal structure.

The view that the vascular and renal changes are not merely parts of a general fibrosis is supported by the frequency with other organs likewise amenable failed to share. A general fibrotic tendency would be likely to involve the liver; but there appears to be little association between cirrhosis of the liver and the corresponding change in the kidney. I found that cirrhosis of the liver was present 37 times in 250 cases of granular degeneration of the kidney—a proportion of about 1 in 7 (p. 125); and subsequent writers have been led to an almost identical conclusion. Dr. Grainger Stewart found cirrhosis of the liver in fifteen per cent. of his cases of this renal disease; while Dr. Galabin,[1] in 79 cases of the same class at Guy's Hospital, found cirrhosis of the liver in 10, both these observers thus agreeing with the estimate of 1 in 7 as the proportion of hepatic cirrhosis in cases of granular degeneration of the kidney. And a similar exemption might be established on behalf of other organs—the fibrosis, to say the least, is far from universal.

If the circulatory change be caused by the renal it can scarcely be otherwise than by way of glandular incapacity and consequent impurity of blood. But within this broad statement there are differences of opinion which make further consideration necessary.

Much light has been thrown on this subject by the sphygmograph; and herein I must be indebted chiefly to the labors of those who have devoted themselves especially to this instrument.[2]

Taking first the most marked condition, the pulse, with the advanced granular kidney, differs from that of health chiefly in this—the tension of the vessel and the distention of the whole arterial system are increased. The systolic upstroke is exaggerated, the diastolic downstroke retarded, and increased pressure is needed to bring out the movements of the vessel. And that the vessel is fuller and tighter than natural is as evident to simple touch as with the instrumental recorder. If in a well-marked case the finger be passed to and fro over the wrist the artery will give the impression of a hard, prominent, and unvarying cord, like a tendon rather than a vessel. The constant arterial distention may presumably be due to general excess of blood, in which case the condition would be shared by the veins; or, if limited to the arterial system, either to its being filled too fast or emptied too slow. We must dismiss the first condition. A general plethora or a superabundant total of blood certainly does not declare itself in the appearance of the persons in whom the change in question is most evident. Nor is there with them any febrile action or cardiac quickening to drive it into the arteries faster than it should flow out. We must find the cause of the repletion in slow emptying of the arterial system, and look for obstruction of its channels of exit, either in the smaller arteries or the capillaries; in the former case we may attribute the repletion, with Dr. Johnson, to spasm of the arterioles, in the latter, with Bright, to retardation in the capillaries.

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[1] Op. cit., page 19.

[2] See Sanderson's handbook of the sphygmograph; an inaugural thesis on the connection of Bright's disease with changes in the vascular system, by Dr. Galabin; a paper by Mr. Mahomed on the etiology of Bright's disease, Medico-Chirurgical Transactions for 1874.

Though perhaps it matters little practically whether we place the hinderance in arteriole or capillary, in the smaller vessels or the smallest, yet the distinction is not without interest.

First as to Dr. Johnson's view. This represents the heart and arteries as acting in antagonism, and hypertrophied by the efforts each makes to get the better of the other, the arteries contracting beyond their wont in the endeavor to shut the blood out of the tissues, the heart using increased efforts in the attempt to overpower their resistance and drive it in, and both, like conflicting athletes, increased in muscle by the exercise.[1]

But it may be asked whether this hypothesis, representing the heart and arteries as animated by different and opposed volitions, is consistent with what we know of their action in health. The heart and arteries may with more correctness be represented as confederates than antagonists. They become less separate as we descend the animal scale, until at last they are merged into one organ. There is reason to believe that the heart would be quite inadequate to force the blood through the systemic vessels without help on their part; and indeed the only feasible view of the mechanism of circulation is that the wave of blood which arises in the ventricle is helped on by advancing zone of arterial contraction which follows swiftly in its rear, and by a sort of vermicular action continues the ventricular impulse as far as arterial muscle extends. If this be so in health more or less of the same action probably takes place in disease. A disorder of which the results are common to the whole body, and with which life is long consistent, is not likely to be attended by such inversion of the natural process of circulation as to replace the necessary co-operation of the heart and vessels by a novel and unnatural conflict. Dismissing the stopcock theory, therefore, as improbable, we revert to the older view of capillary hinderance, and in this, as it is scarcely possible to doubt, we find the key of the whole mystery of the cardio-vascular hypertrophy. That the composition of a circulating fluid affects the rate at which it circulates has been recognized since the experiments of Poisenille with artificial solutions; and in the living body the arrest of the blood in the right side of the heart under asphyxia is sufficient indication of the difficulty with which unaërated blood moves through the pulmonary vessels. And in systemic blood it is easy to believe that renal excrementa may have some such retarding effect as in the pulmonic is due to its unoxidized components. Given the capillary resistance the whole problem clears. The heart and the arteries, whose common functions it is, jointly or alternately, to carry on the blood, become habitually distended, are stimulated by distention to overaction, and by overaction to hypertrophy. As both heart and arteries are exposed to the same causes, both are affected in the same manner and at the same time. But, in addition to the hypertrophic action, the blood-pressure upon the interior of the arterial system, and possibly also the irritant effect of the contaminated blood, produce alterations of a degenerative or subinflammatory kind.[1] Congestion, or increased blood-pressure, causes fibrosis in vessel-walls, as elsewhere. And the degenerative process is abundantly witnessed by the state of the muscular nuclei and of the inner layer of the muscular coat.

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[1] To do justice to this view I must add that it has recently received support from some ophthalmoscopic observations reported by Dr. Gowers to the British Medical Association, at the Sheffield meeting, August, 1876. In cases where the heart has become hypertrophied and the pulse hard, as the result of albuminuria, he represents the retinal arteries as visibly narrowed. as if by muscular contraction. Should this observation be confirmed, and the condition found to be of regular occurrence in such circumstances, it must influence our conclusions in a manner which as yet it cannot safely do.

The view that the cardiac hypertrophy is not caused by the arterial thickening, but results in common with it from the capillary obstruction, is supported, not only by the development of the cardiac hypertrophy often before the corresponding change in the arteries is distinguishable, but also by the general absence of the cardiac alteration in lardaceous disease, where the arteries, though not affected in the same way, are at least as much thickened, and apparently as much obstructed, as in the other forms of albuminuria.

Gathering together the conclusions from what has been advanced, they are as follows:

The arterial thickening of renal disease is due to increase of muscle, together with degenerative and fibroid changes.

A true muscular hypertrophy occurs in the arteries, as in the heart, in consequence of the same hinderance in front of both; superadded to which there are changes in the vessel-walls due to increased pressure by the blood and the irritation by its morbid constituents.

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[1] Atheroma, due in the first instance to cell-proliferation, akin to inflammation, is rarely absent in these cases, however young the subject. In almost all the instances of fatal albuminuria, given earlier in this chapter, specks of atheroma were discovered on the mitral valve or the arch of the aorta.

The cardio-vascular thickening is almost invariable with the granular kidney; some degree of it is commonly to be found as the result of the more lingering forms of nephritis; and it has been traced to destruction of renal tissue by stone or other accidents obviously unconnected with general fibrosis.

Though more or less exaggeration of the renal fibrous tissue is generally to be detected as the associate of the cardiac and vascular change, insomuch that simple hypertrophy of the heart may commonly be taken as a sign of renal fibrosis, yet the circumstances in which the renal nucleation occurs are often such as to show that it is the result of prolonged morbid action proper to the gland itself, and not of any tendency common to the whole body or shared by other organs. The renal fibrosis is obviously of local origin when a result of ordinary nephritis; and a similar inference may be drawn when the kidneys have become granular in childhood, in consequence of some local condition, the diffusion of inflammation at first catarrhal, or the action of retained urine.

Fibrosis of the kidney is only, in an insignificant proportion of cases, associated with a similar change in the liver, as if these two organs were generally acted upon separately rather than by a common influence.

Hypertrophy of the left ventricle arises simultaneously with the vascular change, or, if there be a difference of date, rather before it, the cardiac apparently not so much a consequence of the vascular change as of common origin with it.

The cardio-vascular change is consequent upon renal inefficiency, and is apt to be found in all disorders whereby the renal excreta are much and for long diminished.

The vascular thickening and the cardiac hypertrophy are both due to capillary resistance and arterial distention, and are consequences rather of renal inefficiency, however produced, than belonging to any definite type of renal disease.

The relations of the lardaceous disease to the cardio-vascular hypertrophy, which have been sufficiently considered in a preceding chapter (page 193), fall in with the views which have been expressed as to the circumstances which immediately determine the hypertrophic process.

The usual exemption of lardaceous disease from the cardio-vascular complication may be due, first, to the fact that in this disease the products of renal excretion are, until near the close, little if at all lessened, as witnessed by the small liability to uræmia which it involves; and, secondly, to the relief to arterial pressure which is afforded by the exhausting discharges with which this disorder is so constantly associated. The fact that with lardaceous disease, notwithstanding the usual absence of cardiac hypertrophy, the arteries are thickened—not, indeed, in quite the same way as with other forms of renal disease, but very generally, and in a manner which would seem as obstructive—is enough to show that the exaggeration of the ventricle is due to something more than mere vascular thickening.

But cases occur as a constant minority in which lardaceous renal disease, when of long standing, is followed by the whole cardio-vascular series, even to retinal and intra-cranial hemorrhage. And when it is considered that as lardaceous disease continues it is apt to engender both tubal and interstitial nephritis, while it is possible that the relieving discharges may not have occurred or may have ceased, it is sufficiently obvious that whatever changes may be among the issues of other forms of renal disease must in certain cases ensue upon this.

CHAPTER XV.

ON THE RETINAL CHANGES COMMON TO ALBUMINURIA.

As a corollary to the preceding chapter I will proceed to describe in brief some of the visual changes which occur in the course of renal disease, with especial reference to what has been termed albuminuric retinitis.

Imperfect or complete amaurosis, dimness of sight, or total blindness have long been known in connection with Bright's disease. The ophthalmoscope has taken these disorders for the most part out of the category of the functional and provided them with name and local habitation as definite organic lesions; while more recent observations have shown that, though local, they are not isolated, but are only the outposts of the wide system of arterial change of which I have endeavored to explain the relations.

Before proceeding to visible disease it may be well to state that there is a form of transient amaurosis which comes on apparently as a direct result of uræmia, and is not accompanied by any organic changes such as the ophthalmoscope can detect. This, which has been well described by Dr. Clifford Allbutt, amounts to swimming and indistinctness of vision, or various degrees of dimness, such as to efface the distinction of objects, or even to cause transient darkness. This impairment of sight comes on and usually passes off with vomiting, headache, convulsion, or other manifestation of cerebral uræmia. It may complicate more permanent and visible disease, but is essentially independent of it. It would seem to depend upon some similar change, whether to be called vascular or toxic, to that which produces other nervous symptoms to which the term uræmic is applied.

Passing to the more tangible disorders of the eye which concur with albuminuria, and which depend upon alterations in vascular tension or arterial structure, the retina is subject, as the ophthalmoscope has made known to us, to a complication of disease characterized by hyperæmia, serous infiltration, hemorrhage, and, sooner or later, striking white spots, apparently of fatty degeneration. The changes, though continually intermixed, may occur separately, and be classed as of three kinds:

The first is mere retinal effusion, or œdema, which gives a diffuse milkiness or opalescence to the fundus and an indistinct outline to the disk.

This is the diffuse infiltration of Graefe. It occurs apparently as the result of the increased vascular tension which may either belong to permanent granulation of the kidney or be a temporary accompaniment of passing disorder. It varies with the state of the circulation, is amenable to treatment, especially to the action of purgatives, and is susceptible of recovery when the kidneys are so. It begins somewhat gradually, and is productive, not of darkness, but mist.

This derangement is often succeeded by or associated with the others—hemorrhagic patches and white spots, the two often occurring together, and creating a condition if not pathognomonic, at least very characteristic of albuminuria.

The hemorrhages are analogous in their mode of formation to those which occur in the brain; they commonly affect the subjects of advanced though not necessarily obtrusive granular disease and the characteristic cardio-vascular change; they are apt to break out suddenly after the manner of hemorrhage, to affect both eyes, though neither symmetrically nor quite simultaneously, and may be large enough to cause extensive darkening of the field of vision. The spots, however, are more often small and numerous. They are apt to collect around the disk and along the arteries.

The white commonly associated with the hemorrhagic spots are found about the optic disk or yellow spot, and appear to be patches of fatty deposit or degeneration. They are usually white or cream-colored, reflect light strongly, and occupy, as is shown by their concealing the blood-vessels, the anterior layer of the retina. These have been thought to be a late stage of the blood-spots, and no doubt retinal hemorrhages in their removal whiten; but the patches in question would seem more often to be of fatty change, dependent on arterial degeneration, but unconnected with extravasation, at least of blood in its entirety.

To such evidence as is afforded by the ophthalmoscope it may be added that examination of the eye after death shows that, in addition to the white and hemorrhagic spots which have been described, there is more or less serous infiltration of the optic nerve and disk; while the connective tissue of the retina is increased, the arteries thickened and often atheromatous, and the capillaries dilated.[1]

More or fewer of these alterations, which though of different kinds alike depend on increased arterial tension and the vascular changes which are associated with it, are commonly mingled in the same case, and make it convenient to place together their clinical results in a common description.

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[1] For illustrations and further description of the state of the eyes produced by albuminuria I may refer to an admirable plate in Liebreich's Atlas; to a paper by Mr. Hulke in the Ophthalmic Hospital Reports for January, 1866; to Power's Illustrations of Diseases of the Eye; Allbutt's work on the Ophthalmoscope; and Brudenell Carter on Diseases of the Eye. To Dr. Allbutt, in whose hands the ophthalmoscope becomes an instrument of medical diagnosis, the physician owes especial acknowledgment.

As regards the function of sight the retinal affection of albuminuria, so long as its results are limited to injection and œdema of the retina, presents itself as a mistiness or cloudiness of vision coming upon both eyes, though not necessarily equally or quite simultaneously—not such as to shut out the light or cause total blindness, but enough to obscure the outlines of objects and interpose a fog between the inner organs and the outer world. With the development of spots, whether hemorrhagic or degenerative, the obscuration may take a definite unchanging shape which can be accurately sketched or described—an old gentleman with, as I could not doubt, granular degeneration of long standing, used to represent a crescent moon as a dark figure always before one eye. Large hemorrhage may be so placed as suddenly to darken much of the field of vision; but more often such outbursts, from their size and position, play only a concurrent part in the impairment of sight. Increasing dark spots and deepening mists may at last, or even early and abruptly, cause what practically amounts to blindness so far as the distinction of objects is concerned, though seldom amounting to the total exclusion of light.

The state of vision in these cases often fluctuates; I have known nearly total blindness, with retinal change of corresponding extent, consequent upon the albuminuria of pregnancy, to improve by mere lapse of time until the subject of it, who had been quite unable to distinguish objects, could see her way about. And in other circumstances, even though connected with chronic and hopeless renal disease, the retinal affection will lessen, in a striking manner, under measures which reduce the tension of the pulse. Drugs which act first upon the bowels

> "—— from thick films shall purge the visual ray."

Under calomel and jalap-powder occasionally, with the sulphate of potash or magnesia habitually, the clouds will lighten much as the hardness of the pulse, the nausea, and the headache diminish. But perhaps improvement in such cases is all that can be hoped for; cure is as difficult as to the vascular and renal state on which the retinal lesion depends.

As to the subjects of the retinal disease they are usually either persons who, whether old or young, have advanced granular kidneys and hypertrophied hearts; or they are women who have become albuminuric with pregnancy. With the first class it often happens that the visual impairment is the first to attract notice. The patient may think himself, save as regards his sight, in good health; he may have no dropsy and but a trace of albumen; but examination will almost certainly show that all the links of the morbid chain, the renal, the arterial, and the cardiac, have been fully forged. With pregnancy the renal disease is presumably of the same type, though often of recent date. Albuminuria of this origin is remarkable for the frequency and severity of the retinal affections with which it is accompanied.

Whether the retinal is ever associated with any other than the granular or fibrotic kidney is a question of large interest, as bearing upon the connection of renal with cardio-vascular change.

It may be briefly stated that though most common with the granular kidney there is no type of renal disease which may not acquire the albuminuric retina. This visual change, however, like that of the cardio-vascular series, is an index of chronicity, and may, in most instances, be accepted as a sign that fibrotic renal changes have at least begun. With the granular kidney of every date and kind, perhaps most of all with that of pregnancy, the retinal complication is common. It is so much more common, indeed, with the granular than with any other type of kidney, that its occurrence in this association is the rule, in any other an exception. Instances have been recorded (Dodd and Smith, page 229) in which the retinæ have been characteristically affected, even in childhood, when this renal disease has existed.

And with persistent albuminuria as the result of scarlatinal or other nephritis, in which the kidneys, though possibly not granular, are almost certainly fibrotic, retinal hemorrhage and its attendant deteriorations are by no means unknown. I may refer to the case of Burr, related at page 232, in which these changes occurred in the course of scarlatinal nephritis, the kidneys after death being smooth and enlarged, but with evidence microscopically of early fibrosis. As another example of the same complication of scarlatinal albuminuria I may instance the following:

A girl had scarlatina at the age of three years, after which, though no dropsy ensued, she failed to recover her health, but remained ailing and languid. When eight, she began to suffer from vomiting and headache, together with abdominal pain, and so continued to do at intervals until eight months later. She was brought to the hospital in a marked state of chronic Bright's disease, connected, as it was not possible to doubt, with granular kidneys. She had no dropsy. The urine was pale, generally excessive in quantity, fishy in smell, albuminous to a quarter, and abounding in large coarse granular casts. She suffered much from headache and vomiting, and had occasional attacks of uræmic drowsiness. The heart was hypertrophied, the temporal and other superficial arteries tortuous and hard. With the ophthalmoscope both eyes showed the results of extensive hemorrhage around the disks.

I could instance not a few similar examples where characteristic retinal changes have ensued upon scarlatinal albuminuria, where the albuminuria has been persistent, the heart hypertrophied, and the retinal a late complication. Probably in all such, if we may judge by observations in cases which have reached their close, interstitial fibrosis existed, not as any part of a constitutional change, but as one of the results of the glandular inflammation ensuent upon the febrile disorder.

Making search among the recorded experience of others, I find many instances of the same sort in which the characteristic retinal change has followed upon scarlatinal disease, though the purely tubal character of the disorder has not been certified by recovery or the absence of interstitial fibrosis by the microscope. The large white smooth kidney has been often found after death[1] in these cases, but the heart has usually been hypertrophied, and no doubt secondary fibrosis developed.

With nephritis, which is wholly catarrhal—or, from the fact of recovery, has been presumed to have been so—retinal spots and hemorrhages appear to be of possible though of extremely rare occurrence. Dr. Clifford Allbutt tells me that he has seen at least three cases of optic neuritis with scarlatinal dropsy in which recovery has ensued in both respects. This retinal change, therefore, may result from purely tubal disturbance; and however more frequent when the organic disorder has transcended these narrow limits, it has no necessary connection with fibrosis.

In witness of the infrequency of such retinal conditions with passing renal disease, I can adduce, from the Hospital for Sick Children, a multitude of cases of temporary and therefore tubal nephritis sequent, for the most part, upon scarlatina, without the observation in a single instance of either retinal change or disturbance of sight to suggest it. My point of view, however, is more renal than retinal; it is possible that an observer with special skill might have detected minor changes which in these instances escaped notice.

As to the retinal with the lardaceous disorder some examples are to be found, just as hypertrophy of the heart, however rarely, occurs sometimes in connection with that renal condition. A most characteristic hemorrhagic retina was in one case under my own observation associated with very advanced renal disease undoubtedly of this character. And other instances of the same concurrence are on record. Dr. Grainger Stewart mentions one in which the renal disorder had lasted ten years; one is quoted by Dr. Allbutt, in which the origin in suppuration was sufficiently characteristic of the disease, where not only the albuminuric retina but hypertrophy of the heart existed. Such examples, of which more might be adduced, show that occasionally, though exceptionally, the optic change accompanies renal disorder of the lardaceous type. But, looking at the chronicity of the cases in which this concurrence has been recognized, and at the fact that more or less renal fibrosis is a common associate of advanced lardaceous disease, it cannot but be thought likely that this secondary alteration was present in each.

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[1] Several such cases are recorded in Dr. Allbutt's work, already referred to. An instance of albuminuric retinitis, with "the inflammatory form" of Bright's disease which would probably come under the same category, is alluded to by Dr. Grainger Stewart. Edit. 2, page 97.

In conclusion, retinal hemorrhage and maculation, which are but results of the cardio-vascular change, are distributed similarly to it. Although the granular kidney coexists with them in so large a majority of instances that the retinal may be taken with little error as diagnostic of that renal condition, yet there are sufficiently numerous exceptions to show that the retinal may be a late consequence, either of tubal or diffuse nephritis or of the lardaceous change. It seldom occurs independently of renal fibrosis, because a measure of renal fibrosis is a frequent if not a necessary superaddition to chronic disease of either of the other kinds; but there is evidence that it does so occasionally; and that the optic manifestation is often the issue of disease, which in its origin was not common to the arteries but local to the gland, is certain.

The evidence points with sureness to the conclusion that the retinal, like the arterial change, is not associated with any specific renal condition, but is the issue direct, or indirect, of the increased arterial tension, which loss of renal function in many shapes may entail.

CHAPTER XVI.

THE BLOOD IN ALBUMINURIA.

WITH our present information we are not able to make such distinctions between the condition of the blood in each of the forms of renal disease as the difference in the symptoms would lead us to expect. Our knowledge of the blood is but rudimentary, and is much less complete than our knowledge of urine. It is probable that in many cases the condition of the blood may be best arrived at by assuming that it varies inversely with that secretion. This holds good so far as the changes in the urine are produced by renal disease. Whichever of the urinary elements is absent or deficient in quantity is present in excess in the blood. If the urine contain anything which it should not, or has any of its constituents superabundant, the blood is robbed to a corresponding extent.

Observations as to the state of the blood were made by Dr. Bostock in some of Bright's original cases, and soon afterward by Dr. Christison[1] and Dr. Owen Rees. Analyses have since accumulated, but comparatively little has been added to what was thus early ascertained.

Generally speaking, the changes which are caused in the blood by albuminuria are these.

The albumen and the corpuscles are diminished. The water, the fibrin, the salts, the creatine, creatinine, and extractives are increased. Urea and uric acid are superadded. These statements hold good whether the kidneys be affected by nephritis, granular degeneration, or the lardaceous change.

Taking the subject more in detail, and using only cases where the nature of the renal disease was ascertained by *post-mortem* examination, or was placed beyond doubt by other circumstances, the following particulars may be given with regard to each of these forms of renal disease. The numerical results of the analyses referred to, to save space, are arranged in a tabular form.

NEPHRITIS.

In a fatal case of scarlatinal dropsy, given by Dr. Christison,[2] the blood was analyzed about three months after the commencement of the fever, with the following results. At the time of the examination the urine was increased in quantity. "The blood presented a thin buffy coat, a very small crassamentum, an abundant slightly lactescent serum of the high density 1031." The corpuscles were diminished to a third of their proper amount, the fibrin and water increased. In this case, as in others, Dr. Christison considered the milkiness of the serum to indicate the presence of oil.

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[1] Granular Degeneration of the Kidneys. 1839.

[2] Dr. Christison on Granular Degeneration of the Kidneys. Appendix. Case 10.

Frerichs has also found fat in considerable quantity in the serum under similar circumstances.

Dr. Hassall[1] has examined the blood in a fatal case in which, although no *post-mortem* was obtained, it is evident, from the symptoms, that tubal nephritis existed, with fatty change in the epithelium. The albumen was found to be diminished to about a third of its normal amount.

Urea has been found in the blood,[2] and in the cerebro-spinal fluid in patients who have had the disease in question—once as the result of cholera, once of typhus.

I obtained a considerable quantity of uric acid from the brain-tissue of a young woman who had died of convulsions consequent upon scarlatinal albuminuria. The kidneys were large, smooth, and injected.[3]

GRANULAR DEGENERATION.

The analyses of the blood in this disease will show the truth of the general statements which have been made.

Dr. Bright gives the analysis of the blood, as made by Dr. Owen Rees, in two fatal cases of granular degeneration.[4] Both were characteristic examples of the disease, and were attested by *post-mortem* examination.

These observations accord with others in the diminution of albumen and corpuscles, but not as regards the apparent diminution of salts.

Dr. Hassall has given an analysis of the blood in the case of albuminuria associated with gout, which from the particulars he gives may be looked upon as certainly one of granular degeneration. The water was increased, the albumen was diminished. The fibrin and corpuscles, taken together, were diminished.[5]

Dr. Harley has given elaborate analysis of the blood from a case of the albuminuria of pregnancy, in which the blood was examined during the presence of dropsy and albuminuria previous to delivery, and again after delivery, when the patient was in apparent health.[1]

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[1] Lancet, 1864. Case of Edward G.

[2] Lehmann's Phys. Chemistry. Cavendish Society, vol. i., p. 165. See Path. Trans., vol. xviii. Case by Dr. Cayley.

[3] Path. Trans., vol. xviii.

[4] Cases of Charles Scott and Wm. Curtin, by Drs. Bright and Owen Rees. Guy's Hosp. Reports, 1843, pp. 196 and 209.

[5] Lancet, Dec. 17, 1864. Case of Francis S.

The comparison shows that, during the disease, water and inorganic salts were increased, the albumen and corpuscles diminished, the fibrin not much altered, but diminished to a slight extent.

Speaking generally, it may be said that with granular degeneration the blood does not become so much impoverished as with the more acute disorder, but that it equally abounds with urea, and probably with other excrementitious matter.

LARDACEOUS DISEASE.

The readiness and certainty with which this disease can be recognized enables us to turn to account some of the cases of the earlier observers, and discover a significance in their writings which was hidden from themselves.

Dr. Bright reports a case of albuminuria complicated with phthisis and ulceration of the bowels, in which the waxy condition of the spleen, liver, and kidneys was clearly described, though not by name.[2] The loss of albumen and corpuscles is considerable. It is to be regretted that the fibrin was not estimated separately, but Dr. Christison has, to a certain extent, supplied the deficiency. He gives the case of a lad 16 years of age, who presented the symptoms of the disease in a very marked way, though it is not clear what the primary disorder was. Thirst, frequent micturition, and œdema, were succeeded by headache, insensibility, and convulsions. The head-symptoms were apparently coincident with suppression or great diminution of urine. During the last two days of his life he was bled to 60 ounces.[3]

The *post-mortem* examination showed extensive hepatization of the lungs. The liver was somewhat translucent, the spleen partially of a rose-red, the mucous coat of the bowels "as if soaked with serum," an appearance very characteristic of the lardaceous change. The kidneys were pale, finely granular on their surfaces, marked with stellate vessels ; on section, grayish yellow in the cortical part, of a pale rose color in the cones.

On analysis the serum of the blood had a density as low as 1019. The blood-globules and the solids of the serum were greatly reduced, the water was greatly increased ; the fibrin was increased to nearly double its proper amount.

The blood contained a large quantity of urea. In this case, the extreme poverty of the blood must be attributed, in some measure, to the remorseless bloodletting. In connection with the increased amount of fibrin, it may be mentioned that the heart and large vessels were found to contain very large and tenacious coagula.

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[1] Harley on Albuminuria, p. 37.

[2] Case of James Back. Guy's Hospital Reports, 1843.

[3] Dr. Christison, loc. cit., p. 194. Case 7.

Analyses of Blood in Albuminuria—Proportions in 1,000 parts.

Case.	Water.	Corpuscles.	Fibrin.	Corpuscles and fibrin together.	In 1,000 parts of serum.			
					Solid matter.	Albumen.	Salts (inorganic).	Urea.
Average Composition of healthy Blood according to—								
Christison	775.7	137.1	3.8	140.9	83.4	—	—	
Owen Rees	792.2	—	—	119.95	87.85	79.5	7.5 (alk.)	
Hassall	787.6	—	—	143.0	—	69.4	—	
In Nephritis—								
1. Case 10, p. 440. After Scarlatina. Dr. Christison	857.2	42.7	4.5	47.2	95.6	—	—	
2. Lancet, 1864. Edw. G. With fatty change. Dr. Hassall	889.5	—	—	86.8	—	23.7	—	
Frerichs. Scarlatinal dropsy. Youth, aged 18	908.10	130.7	3.4	131.4	91.9	81.4	9.09	
" " " Girl, aged 21	938.9	122.0	4.2	126.2	61.1	51.7	—	
In Granular Degeneration—								
3. Dr. O. Rees. Guy's H. R., 1843. Wm. Curtin	853.11	—	—	65.61	81.28	68.5	6.0 (alk.)	.05
4. " " " Chas. Scott	835.85	—	—	81.61	82.52	—	—	
5. Dr. Hassall. Lancet, 1864. Francis S.	821.0	—	—	120.0	—	59.0	—	
6. { Dr. Harley. Albra. of pregnancy. Before delivery.	825.9	103.51	2.30	105.81	72.68	60.69	9.08	
6. { " (patient convalescent). After "	768.7	141.7	2.85	144.55	94.0	86.5	5.000	
In Lardaceous Disease—								
7. Dr. Christison, p. 426. Case 7	885.3	56.4	6.2	62.6	52.1	—	—	
8. Dr. Owen Rees. James Back. Guy's H. R., 1843	828.9	—	—	94.1	76.98	65.15	—	

Blood Corpuscles in Renal Disease as Estimated Numerically.

Until of late there has been no satisfactory way of estimating the corpuscles of blood; but now the hæmatometer of Hayem and Nachet[1] provides a means which is at once simple and accurate of ascertaining at least their number in a given measure of blood. I need not here describe the process further than to say that it consists in counting with the microscope the corpuscles contained in a given measure of a definite dilution of blood. The blood, as drawn from the finger, is measured in a capillary tube mixed with a certain proportion of a saline substitute for serum, and placed in a cell accurately adapted to contain a layer of one-fifth of a millimetre in thickness. Upon this if one-fifth of a millimetre square be marked out superficially by means of a micrometer eyepiece, it is obvious that this figure, when looked at vertically, will mark the limits of a cube of corresponding dimensions. In this, after subsidence, the corpuscles of both kinds can be accurately counted, and by taking the average of several observations—I have usually counted the red in six different places, the white in twenty—a fair estimate of the number of corpuscles of each sort in the cubic bulk of the mixture can be formed. Multiplication will give their number in any required quantity of undiluted blood. I have preferred to record the numbers as observed, as more easy to deal with and compare while unswollen by arithmetic; but should the reader desire to enumerate the corpuscles of either sort in a cubic millimetre of unmixed blood he has but to multiply by 31,375. The cell and other apparatus used in the experiments were the same for all, and the blood was drawn in each instance about three hours after the hospital dinner-time—all the diseased subjects were hospital patients—so as to lessen, as far as practicable, the variation due to time of food.

The total number of corpuscles proper to vigorous health and adult life—and for childhood the number does not seem to be very different—is, according to the observations both of Hayem and Keyes, about five millions in the cubic millimetre of blood, or 159 in the cube of dilution examined. It will be seen that the cases I have examined as presenting the conditions of health have not departed much from this standard—though somewhat higher, 166 as the average, against 159. It is obvious that in spite of every care there must always be minute differences in the capacity of the cells used by different observers.

The observations show first that with every kind of albuminuria there is an extraordinary reduction in the number of red corpuscles, together with some increase in the white, not only relatively to the red, but in proportion to the measure of blood. The loss of red corpuscles appears to be greatest with the more persistent forms of disease, notably with the granular kidney, in one case of which they were nearly reduced to half the average of health. With nephritis, though the loss was generally less, it was in some instances fully as great.

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[1] The mechanical arrangement I have made use of is that described by M. G. Hayem, Gazette Hebdomadaire, 1875, p. 291. I have been indebted also to a valuable paper relating to the use of the same apparatus by Dr. Keyes, of New York, in the American Journal of Medical Sciences for January, 1876.

With regard to lardaceous disease and its antecedent suppuration these observations have especial interest.

As to suppuration, in some instances in which this process had proceeded to the obvious exhaustion of the patient, the corpuscles, in a given measure of blood, were more than naturally numerous, as if the fluid part had wasted more than the corpuscular. Altogether the diminution of corpuscles under this discharge was less than might have been expected ; and more strangely still, the white were generally increased, whether regarded in proportion to the red or to the bulk of blood. With our present knowledge it is, perhaps, better to be content with simply recording the fact than to attempt to explain it.

Passing to the established lardaceous disease, the diminution of red corpuscles, though decided, is smaller than with other forms of albuminuria ; and similarly the increase of white, though evident in every aspect, is less marked than in other circumstances of renal disease. An instance of lardaceous disease in a state of retrogression—in which we may infer an opposite state of blood to those in which the disorder is progressing—concludes the series. The red corpuscles were numerous, the white few. A diminution of red and an increase of white appear to be the characteristics of the advancing disease ; though as neither alteration is greater than often occurs where no lardaceous disease exists, we cannot attribute especial importance to these deviations.

For comparison with those instances of lardaceous disease in which the spleen was enlarged, I have appended several observations upon cases of rickety enlargement of this organ ; in both classes it is evident that the white corpuscles are increased, though in neither to any greater extent than is often due to simple renal anæmia, be its organic association what it may. In whatever aspect we regard it, the corpuscular loss of lardaceous disease displays the dimensions rather of a secondary consequence of renal disease than as a process essentially concerned in its development.

In introducing the tables, I may state that the number assigned to the red corpuscles in a square is the average of several countings, usually of six. With regard to the white, since from their usually small number the average in one square cannot be expressed as a whole number, I have stated it as a fraction, of which the denominator gives the number of squares counted, usually twenty. The averages at the end of each table give that of the red for one square, that of the white for twenty.

Number of Corpuscles in $\frac{1}{31,375}$ of a cubic millimetre of Blood in Health.

Name.	Age.	Case.	Total.	Red.	White.	White to red 1 to —.	Appearance of corpuscles.
Male............	23	Robust health—just returned from a tour in Norway..	—	185	$\frac{8}{20}$	462	Natural.
"	26	In vigorous health—on duty in hospital............	—	168	$\frac{8}{20}$	420	"
"	9	Apparently in perfect health......................	—	170	$\frac{5}{20}$	680	"
Female....... ...	14	Apparently in perfect health—just back from the sea-side..	—	157	$\frac{9}{20}$	348	"
"	6	Apparently in perfect health......................	—	165	$\frac{9}{20}$	366	"
"	19	A hospital nurse, apparently in perfect health, but thought to be delicate.........................	—	144	$\frac{7}{20}$	411	"
		Average of health........................	—	166	8 in 20 squares.	448	

Number of Corpuscles in $\frac{1}{31,375}$ of a cubic millimetre of Blood. Tubal or Diffuse Nephritis.[1]

Name.	Age.	Case.	Total.	Red.	White.	White to red 1 to —.	Appearance of corpuscles.
Richard Mumford.	17	Scarlatinal dropsy with empyema, for which aspiration had been repeatedly performed with removal of large quantities of pus. Blood in urine. Extreme anæmia and exhaustion..............................	80	—	—	—	
Henry Cook......	22 months.	Scarlatinal dropsy—dropsy 8 weeks, rash 11 weeks. Much apparent anæmia, but now no œdema. Little albumen in urine. Child improving	—	94	$\frac{14}{19}$	184	
John Bowler......	23	Acute general dropsy of 5 weeks' duration. Patient stout and somewhat florid in appearance. A cellarman who has drank freely of wine. Urine black with blood. (Under Dr. Cavafy).................	—	171	$\frac{3}{20}$	1,140	Corpuscles natural; red well colored.

2d observation..		9 days later. Urine still bloody. Aspect of patient still plethoric.	—	166	$\frac{10}{20}$	332	Corpuscles natural; red well colored.
George Child.....	26	Enormous general dropsy of 1 year's standing, involving cellular tissue and serous cavities. Patient pale. Albumen = $\frac{1}{2}$.	—	116	$\frac{13}{20}$	178	Red natural, white small.
2d observation..		6 days later. Condition much the same.	—	124	$\frac{12}{20}$	206	Red natural, white small.
		Averages.	—	125	10 in 20 squares.	—	

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[1] In calculating the averages, where the red and white are not distinguished the total is taken as red.

Number of Corpuscles in $\frac{1}{31,378}$ of a cubic millimetre of Blood. Granular Kidney.

Name.	Age.	Case.	Total.	Red.	White.	White to red 1 to —.	Appearance of corpuscles.
Walter Kimm....	32	A painter. Marked symptoms of granular kidney. Pericarditis. Much pallor, only a trace of œdema. Died, but no pain.	105	—	—	—	
Richard Hart....	56	Much œdema of very long standing, connected with granulating and contracting kidney, apparently the result of old nephritis. P. M.	95	—	—	—	
Thomas Mason...	47	Exposed to lead. Ill 10 months. Considerable dropsy. Tongue dry, pulse very weak. Died day after examination. Granular kidneys and hypertrophied heart. (Under Dr. Cavafy.)	—	127	$\frac{33}{20}$	77	Red well colored, white small.
Anne Buckley ...	50	Slight œdema for 2 months. Scarlatina 9 months ago. Pale pigmented face. Urine pale, copious; albumen = $\frac{1}{2}$. Old granular kidney?	—	87	$\frac{16}{20}$	108	Red pale, white small.
		Averages	—	103	24 in 20 squares.	—	

Number of Corpuscles in $\frac{1}{31,378}$ of a cubic millimetre of Blood under Suppuration without obvious Lardaceous Disease.

Name.	Age.	Case.	Total.	Red.	White.	White to red 1 to —.	Appearance of corpuscles.
George Smith....	7	Empyema 10 months. Tapped 3 times with total discharge of about 12 ounces of pus; some also discharged by bronchial tubes. Chest shrunk, but general health unaffected.	—	143	$\frac{2}{6}$	429	Natural.
Luke Andrews...	28	Empyema, intermittent discharge for 5 years; for 11 months constant, about half-pint daily by mouth. Aspect of patient that of health........................	—	149	$\frac{7}{5}$	100	"
Frank Stone.....	20	Under Mr. Pollock. Empyema 7 years. Growth stunted, emaciation, much distortion of chest, fingers clubbed. Quarter-pint discharged daily 3 years ago, but now discharge trifling..................................	187	—	—	—	
" "		Later observation................................	—	185	$\frac{7}{2}$	53	
Benjamin Rudd..	5	Under Mr. T. Smith. Scrofulous disease of knee discharging about a drachm a day for 4 months. General health apparently unaffected........................	—	140	$\frac{6}{5}$	116	
Henry Rose......	8	Under Mr. Rouse. Disease of pelvis and hip. Discharge of 4 or 5 ounces a day for 18 months................	138	—	—	—	
John Palmer.....	8	Under Mr. Marsh. Necrosis of femur, discharging about half-pint daily for 4 months. Much failure of health..	—	158	$\frac{2}{6}$	472	White large and well marked.
Walter Hepden ..	6	Under Mr. Smith. Various sinuses connected with diseased bones, discharging together about 1 ounce a day for 9 months. Failure of health....................	—	205	$\frac{5}{5}$	205	
Ann Pescodd....	9	Under Mr. T. Smith. Necrosis of several bones. Amputation of leg 4 months ago. More or less discharge for 14 months, now only about 3 drachms daily. General health fair...	—	137	$\frac{1}{7}$	965	Red faintly colored, some irregular and small.
		Averages..................................	—	160	22 in 20 squares.	—	

Number of Corpuscles in $\frac{1}{31,378}$ of a cubic millimetre of Blood in cases of Lardaceous Disease.

Name.	Age.	Case.	Total.	Red.	White.	White to red 1 to —.	Appearance of corpuscles.
Charles Bowdly..	26	Disease of hip, abscess discharging about 3 oz. daily for 5 months. Disease of bladder. Bed-sores. Much pallor and exhaustion. Liver and spleen not enlarged, but urine considerably albuminous. Under Mr. Pollock. (P.M. examination. Early lardaceous disease of one kidney, the other destroyed by scrofulous pyelitis.) ...	125	—	—	—	Red irregular in size, white natural.
2d observation.		Ditto	—	126	$\frac{4}{6}$	190	
3d observation.		Ditto	—	135	$\frac{21}{20}$	128	
Edward Bristowe.	11	Discharge for 4 years from sinuses connected with hip. Disease now slight. Complexion very sallow. Liver and spleen much enlarged. Urine highly albuminous.	—	155	$\frac{28}{20}$	110	Red irregularly shrunk and faintly colored, white natural.
2d observation.	—	After 25 days iron and good diet........................	—	102	$\frac{19}{20}$	107	
William Pracey..	10	Disease of tarsus 3 years. Discharge about $\frac{1}{2}$ oz. a day. Liver greatly enlarged, spleen somewhat. Trace of albumen in urine..................................	—	155	$\frac{17}{20}$	182	Red small and very pale, white natural.
2d observation.	—	After 25 days iron and good diet......................	—	149	$\frac{15}{20}$	198	
John Shaughran.	11	Phthisis, with purulent expectorations about 1 oz. daily. Liver greatly enlarged, spleen slightly so. Urine not albuminous..	—	119	$\frac{12}{20}$	198	Red pale and irregular in shape.
2d observation.	—	10 days later. Some pyrexia. T. = 102..............	—	126	$\frac{10}{20}$	252	
Sarah Foster....	6	Phthisis. Angular curvature of spine, no sinuses. Much œdema. Urine highly albuminous..................	—	133	$\frac{20}{20}$	133	Red pale, white natural.
Emily Williams..	11	Congenital syphilis, loss of bone from nose, etc. Old glandular abscesses. Spleen enormously enlarged, liver considerably. Trace of albumen in urine............	—	109	$\frac{12}{20}$	181	Red natural, white small.
2d observation.	—	After a week of iodides of potassium and iron...........	—	104	$\frac{4}{20}$	520	White larger.

Number of Corpuscles in $\frac{1}{31,375}$ of a cubic millimetre of Blood in cases of Lardaceous Disease—(Continued).

Name.	Age.	Case.	Total.	Red.	White.	White to red 1 to —.	Appearance of corpuscles.
William Catlin...	13	Disease of hip and pelvis. Lardaceous disease formerly extreme, now subsiding. No evidence of it now, but enlargement of spleen, which is still great..........	—	181	$\frac{7}{20}$	517	
		Averages including all cases	—	132	14 in 20 squares.	—	
		Averages of progressive cases only—(all but the last)	—	128	15 in 20 squares.	—	

Number of Corpuscles in $\frac{1}{31,375}$ of a cubic millimetre of Blood in cases of Splenic Enlargement, not Lardaceous.

Name.	Age.	Case.	Total.	Red.	White.	White to red 1 to —.	Appearance of corpuscles.
Anne Vincent....	1 yr.	Rickets, with enlarged spleen and liver. Very anæmic in look. Under Dr. Barlow.......................	—	105	$\frac{3}{5}$	169	Blood looked watery.
Boy.............	10 months.	Rickets. Large spleen. Pallid, cachectic, almost chlorotic look. Under Dr. Barlow................	—	70	$\frac{2}{5}$	177	Ditto Ditto.
Samuel How.....	1 yr.	Enlarged spleen from rickets. Glands also somewhat enlarged. Under Dr. Gee.......................	—	102	$\frac{2}{5}$	256	Blood looked watery, child was troublesome, and neither of these estimatives quite satisfactory.
" "	—	Another examination at a week's interval............	149	—	—	—	Ditto Ditto.
Fred. Bryant....	10 months.	Rickets. Spleen greatly enlarged—as low as crest of ilium and to within an inch of umbilicus...........	—	89	$\frac{3}{5}$	145	Blood watery, corpuscles natural.
		Averages..............................	—	103	10 in 20 squares.	—	

The numerical results recorded in the preceding tables may be thus summed up: Taking the standard of health at a total of 5,000,000 of corpuscles in the cubic millimetre of blood, of which 4,988,000 are red, 12,000 white, we find that with the cases of nephritis the red in the same measure of blood averaged 3,921,875 with a minimum of 2,949,250; while the white displayed a decided increase, averaging 15,687.

With the granular kidney the average of red was reduced to 3,231,625, the minimum to 2,729,625, the lowest point reached in the entire series. The white corpuscles under the same disease were increased on an average to 37,650; a larger increase of white, and a larger diminution of red than observed with either of the other renal disorders.

Next as to chronic suppuration without sign of lardaceous disease—the departure from the numbers of health under this influence is as regards the red corpuscles imperceptible or inconstant; possibly the fluid of the blood may be reduced together with its solid constituents so that the proportions are little affected. The mean of the red was 5,020,000 in the cubic millimetre, about that of health; the mean of the white 34,512, a sufficiently obvious increase, as has been already observed.

Passing now to the lardaceous condition, and taking first those cases only—all save that of Catlin—in which the disorder was progressive or stationary, the red corpuscles show a reduction, but not to the extent found in other renal diseases. The observations gave an average for the red corpuscles of 128 in the square, or 4,016,000 in the cubic millimetre; the white $\frac{15}{20}$ to the square, 23,531 to the millimetre. Retrogressive disease in which the state of blood, since under it the deposit lessens instead of being added to, must be presumed to be the opposite of that in which the deposition is in progress, is represented by a solitary case. In this the red corpuscles were somewhat above the average of health, surpassing 5,000,000 to the millimetre, the white fewer than normal, little more than 10,000 in the same volume of blood.

BLOOD IN ALBUMINURIA GENERALLY—URÆMIA.

It would be easy to accumulate a much larger number of blood analyses in albuminuria, but I have preferred to make use only of such as can be associated with a definite condition of kidney. Together with these and the results of corpuscle-counting it will be easy to summarize many particulars with regard to the blood under renal disease, though much still remains to be learned.

We can as yet make no very marked distinction between the blood in each disease, though it is obvious that differences must exist, from the fact that the symptoms which result from the state of blood are different in each case. It is clear that with the granular kidney the blood becomes less watery than as the consequence of nephritis, and it may be surmised, though the evidence is insufficient to prove it, that with the lardaceous disease the fibrin is increased to a more marked degree than with other disorders.

With albuminuria of every kind the red corpuscles are diminished, the white increased.

Urea has been found in the blood with each type of kidney, and we are not as yet able to say that there is any excrementitious matter which belongs to the blood of one disorder rather than that of another.

Changes in the blood, of which some have been described, while perhaps others are as yet unknown to us, are the immediate cause of many of the evils which follow upon albuminuria. The absence of change in the nervous centres themselves, notwithstanding that their function is disturbed or abolished, leads to the inference that it is in the fluids, not in the solid structure, that the alteration takes place. This hypothesis is the more satisfying, since the symptoms present in these cases often bear a great resemblance to the effects of some of the narcotic poisons; for instance, opium and belladonna. Besides the presence of excrementitious matter, the superabundance of water in the blood has been supposed, probably with justice, to play an effective part in the production of the symptoms. This seems to be especially the case when the symptoms are of a convulsive kind, convulsions being known to result from excessive depletion.

The general condition is expressed by the word uræmia, which must be held to imply nothing more specific than that the blood is altered by the presence of materials which in their own shape, or under another guise, ought to have passed out by the kidneys.

Opinions have undergone several changes of late years as to the nature of the renal function; whether the kidney, like a filter or dialyzing apparatus, merely allows of the escape of materials through it in the shape in which they are presented to its action, whether it has an active power of construction, discharging as urea and uric acid matters which reach it in some other shape. The older view, that the essential compounds of the urine are not made in the kidneys but only removed by them, was contradicted by the researches of Zalesky, but has been reaffirmed by those of Gréhant,[1] who has shown, with regard to urea at least, that this substance will accumulate in the blood after the removal of the kidneys just as fast as when the ureters are tied, the glands remaining intact. And the conclusion indicated, that urea has its origin mainly outside the kidney, is consistent with so many other observations, particularly with those which assign a large production of urea to the liver—the estimations by Cyon of the proportion of urea in the blood which enters and that which leaves the liver, and the diminished excretion of this substance by the kidneys under the influence of hepatic disease, yellow atrophy or extensive abscess—that we may adopt it as one to which much probability pertains. How far we may extend the conclusion to uric acid is not certain, but there appears to be little doubt that urea at least is mainly of extrarenal origin. Looking at the large amount of urea which is daily made in health, and if it be chiefly made externally to the kidney there is no reason why much less should be produced under many of the circumstances of renal disease, it is not possible but to attribute important results to the amount of accumulation which the diminished discharge must often lead us to infer. Urea has been taken harmlessly into the stomach and injected into the veins with no result beyond increase of urine, but such experiments have been made upon animals whose healthy kidneys could respond to the call and quickly remove the excess; with the exit closed by disease the result may be very different. The symptoms of uræmia may be in part due either to urea as such, or to matters which have resulted from its decomposition.

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[1] Zalesky, Researches upon Uræmia, Tübingen, 1865. Researches upon the Excretion of Urea, by Nestor Gréhant. Archives de Physiologie, par Brown-Séquard, 1870.

Carbonate of ammonia—the result of the transformation of urea—has been regarded as the source of much mischief. This hypothesis is due to Frerichs. It is known that the ammonia which always exists in the breath is increased in quantity when the kidneys are diseased. It is known, also, that the injection of carbonate of ammonia into a vein causes convulsions and nervous disturbance. But the exhalation of the ammonia by the lungs is so rapid, that a very large quantity may be introduced into the circulation with only temporary results. It cannot be supposed that this substance can be made in the body faster than such experiments show that it passes off in the breath. Ammonia has indeed been obtained from the blood in cases of renal disease, but there is little doubt that it has resulted from changes in that fluid after its removal from the body.[1]

It is probable that the symptoms of blood-poisoning in renal disease are not produced by one chemical substance only, but that many are concerned in the result, perhaps in different measures in different conditions of disease. The toxic symptom which result from a mechanical obstruction of the ureter are widely different from those which ensue when the hinderance is in the kidney tubes, and those of acute nephritis again may be different from those of granular degeneration. It is not likely that the state of blood is quite the same in all. Urea, or the products of its decomposition, uric acid, creatine, creatinine, and a variety of substances under the general name of extractives, may all play the part of retained excreta and display each its own toxic influence. We must also attribute something to the loss of corpuscles and of albumen, as well as to the increase of water. Especial symptoms, such as a proneness to the deposition of fibrin in the vessels, result from an excess of this substance in the blood.

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[1] Hammond on Uræmic Intoxication. American Journal of Medical Sciences, 1861, p. 64.

The symptoms which result from such changes in the blood as have been described, have been sufficiently indicated in the particular account of each disorder. Some of the disturbances which have formerly been assigned to uræmia are now known to depend upon specific local changes. The diarrhœa of the lardaceous disease depends upon alteration in the intestinal vessels; the vomiting and dyspepsia often upon similar alterations in the vessels of the stomach; most of the affections of vision which accompany renal disease upon morbid changes in the retina. But there remain many, and most grave, affections of which the cause lies in the state of the blood.

Of these the most important are the disorders of the nervous system, the coma, and epileptiform attacks by which renal disorders are so often terminated, and the lesser affections, drowsiness, headache, cramps, and convulsive movements, as well as various alterations in the temper and state of mind. Vomiting is often a prominent symptom, and it is said that urea and carbonate of ammonia have been found in the matter discharged from the stomach. Diarrhœa, though it sometimes occurs, is less frequently present, except when associated with the lardaceous change. There is reason to believe that many of the inflammatory disorders which are apt to come on apparently spontaneously in the course of renal disease, result directly from the condition of the blood. It has been shown that the injection of urine or urea into the circulation of animals frequently sets up pleurisy and pericarditis, and it seems that the urinary excreta, when present in the blood, have an irritant effect upon the tissues.

CHAPTER XVII.

GENERAL COMPARISON OF THE THREE FORMS OF RENAL DISEASE WHICH HAVE BEEN CONSIDERED.

It may be worth while to place in apposition some of the details which are spread over the preceding pages. A table relating to the ages selected by each disease, and another, showing the symptoms proper to each, have been compiled from the particulars which have been already given.

The following table and diagram (p. 266) show the distribution of each form of disease among the decennial periods of life.

It is seen that nephritis is a disease of youth causing most deaths in the first decade coincidently with the prevalence of scarlatina; many in the third, when the toils and exposures of active life are perhaps most prolific of evil. Granular degeneration, all but unknown where nephritis is most prevalent, belongs to middle and advancing life, and is most fatal between fifty and sixty. The one flourishes upon the febrile accidents of childhood and the susceptibility of youth; the other develops when the habits of life begin to tell and the effects of age to appear. The lardaceous disorder has little to do with either extreme of the mortal course; it has no relation either with the especial disorders of childhood or the deteriorations of declining life, but is chiefly associated with the vices of early maturity and with tubercle and struma, disorders more incident to the young than the old, and in their suppurative form to youth rather than childhood.

The table on p. 266 shows, in numerical detail, the clinical distinctions which separate the three diseases, which it is the object of the present work to distinguish. All the numbers given are necessarily below the truth in proportion as the symptoms which they represent are liable to evade notice.

Briefly reviewing the more important details, it is seen that hæmaturia and pain in the loins belong more especially to nephritis.

Dropsical affections and their immediate consequences are by far the most frequent with nephritis; while they appear less common with the lardaceous disease than with granular degeneration. With regard, however, to the lardaceous change, it must be considered that many of the cases upon which the table is based terminated, as far as the kidneys were concerned, prematurely. Death was in not a few instances due mainly to the affection—phthisic or caries—from which the disease had sprung, and

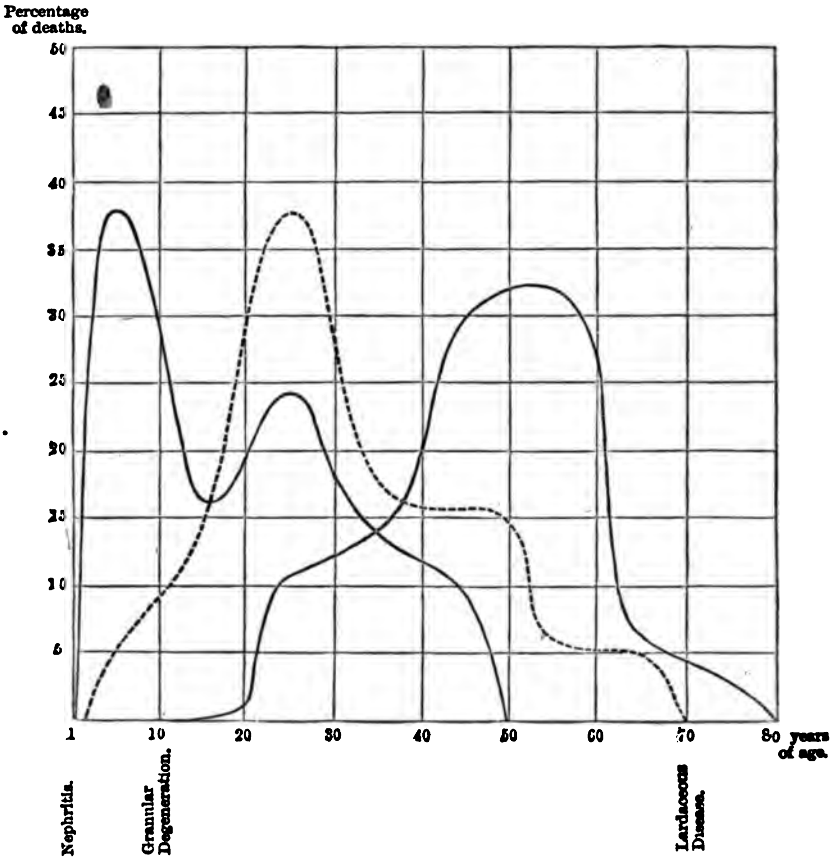
AGE, IN RELATION TO RENAL DISEASE.

Table showing Age at which the three forms of Albuminuria terminate fatally, calculated from cases under the observation of the Author.

PERCENTAGE OF CASES FATAL AT DIFFERENT PERIODS.

Age.	Tubal nephritis.	Granular degeneration.	Lardaceous disease.
From 0 to 10	37	0	5
" 11 to 20	17	1	18
" 21 to 30	23	11	35
" 31 to 40	13	18	16
" 41 to 50	10	30	16
" 51 to 60	0	32	5
" 61 to 70	0	6	5
" 71 to 80	0	2	0

Diagram constructed from the preceding Table, showing the mortality produced by each Disease at the different Periods of Life—Each Decade is represented by a vertical column, the Mortality by the height of the curve.



occurred before the renal symptoms had attained their full development. In cases which are able to progress to their renal ending, it is probable that dropsy is more common than with granular degeneration.

Pneumonia, pleurisy, and peritonitis, more often result from nephritis than from the other varieties of renal disease.

Bronchitis, most common with the granular kidney, is, nevertheless, often associated with the inflammatory. Pericarditis is conspicuously the property of the granular condition; endocarditis or fibrinous deposition upon the valves of the lardaceous.

The tubal affection, with its great liability to dropsical and inflammatory complications, is exempt from the disorders which belong to the circulating system, the more completely the more recent the attack. Hypertrophy of the heart with cardio-vascular thickening, of which it is the sign, is commonly linked with the granular kidney; it is indeed nearly unknown with renal disease unless fibrosis at least takes a part; or, in other words, does not occur but with disease which is so chronic as to have become thus complicated. Hemorrhagic accidents, depending as they do upon structural changes of vessel, are seldom associated with the acute disease. And they are more prone to result from the concurrence of vascular disease, high tension, and cardiac hypertrophy, which is the attribute of the granular kidney, than from the special arterial change of the lardaceous, unaccompanied, as it usually is, with either an exaggerated ventricle or distended arteries. With the lardaceous condition there appears to be scarcely any proclivity to cerebral hemorrhage, one of the most mischievous of the tendencies of the granular, while epistaxis is frequent with both. The hemorrhagic disorder of the retina characteristic of albuminuria generally belongs to the granular kidney, though it may ensue upon either of the other types if only they have become associated, as they do sometimes, though rarely, with exaggerated arterial tension and cardio-vascular hypertrophy.

Diarrhœa belongs especially to the lardaceous disorder. It occurs sometimes with nephritis; rarely with granular degeneration. Vomiting is more evenly distributed.

With regard to both these symptoms, they may be associated either with uræmia or with the extension of the lardaceous change to the mucous membrane. When diarrhœa or vomiting is associated with the waxy kidney, the latter alteration has almost invariably happened.

With regard to head-symptoms, the difference between the diseases is striking. Uræmic affections of the nervous system may be looked upon as the natural ending of the two disorders in which the structural change is essentially limited to the kidneys. With granular degeneration a larger proportion of the cases are fatal in this manner than with nephritis, where inflammatory complications so frequently cut short the course of the disease.

There is a difference in the nature of the attacks which each of these two disorders invites. Convulsions are of general occurrence with nephritis, while with the granular kidney, though convulsive seizures frequently happen, there is a still greater liability to simple coma. With the lardaceous disease the tendency to uræmic attacks of either kind is but small.

As to gout, it is pathologically associated only with the granular kidney. If it occurs with either of the other renal diseases, it is an accidental coincidence, a coincidence necessarily rare with simple nephritis, by reason of the youth of most of its subjects.

Table showing the Percentage of Secondary and other Affections associated with the three forms of Albuminuria which have been described. Calculated from the tables already given.

	Nephritis.	Granular degeneration.	Lardaceous kidney.
URINE, ETC.—			
Hæmaturia	46.1	15.0	8.3
Frequency of micturition	10.2	19.7	8.3
Pain in loins	25.6	13.5	10.4
DROPSY, ETC.—			
Œdema	97.4	72.0	68.7
Ascites	51.2	26.4	25.0
Hydrothorax	28.2	33.6	2.0
Fluid in pericardium	2.5	4.8	—
Erysipelatous inflammation	20.5	4.3	6.2
INFLAMMATORY—			
Pneumonia	25.6	10.2	18.7
Pleurisy	20.5	10.2	10.4
Peritonitis	12.8	4.3	8.3
Pericarditis	2.5	23.5	6.2
Endocarditis	—	5.8	2.0
Bronchitis	20.5	35.4	2.0
Enteritis	—	4.3	2.0
Croup and diphtheria	10.2	—	—
CIRCULATING SYSTEM, HEMORRHAGE, ETC.—			
Hypertrophy of heart	—	45.4	2.0
Apoplexy (sanguineous)	—	4.3	—
Epistaxis	—	5.8	4.1
Hematemesis	—	4.3	—
Purpura	2.5	—	4.1
Affection of retina [1]	—	7.3	—
MUCOUS FLUXES—			
Vomiting	23.0	25.0	22.9
Diarrhœa	12.8	2.9	45.8
URÆMIC AFFECTIONS OF BRAIN—			
Convulsions	25.6	16.1	6.2
Coma, without convulsions	5.1	20.5	4.1
Other cerebral symptoms	10.2	19.7	2.0
GOUT	—	23.5	4.1

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[1] It is not to be inferred that the retinal affection never occurs with the nephritic and lardaceous conditions, but only that there chanced to be no example of this concurrence in the series of cases upon the analysis of which this table is founded.

The following statement from Dr. Grainger Stewart gives similar details. In copying it from his work I have ventured to omit what refers to forms of disease which are complicated or indefinite. As it stands it admits of exact comparison with the particulars of the preceding table.

Table showing the chief complications of the different forms of Bright's Disease. (*Dr. Grainger Stewart.*)

			Hypertrophy of heart.			Lungs and bronchi.			Inflammation of serous membranes.						Liver.				Spleen.		Int-stines.		Brain.
									Pericardium.		Pleura.		Peritoneum.										
	Number of cases.	Dropsy.	Total.	Otherwise explained.	Kidney sole cause.	Congestion and œdema.	Pneumonia.	Tubercle.	Total.	Independent of morbus Brightii.	Total.	Independent of morbus Brightii.	Total.	Independent of morbus Brightii.	Fatty	Waxy.	Waxy and fatty.	Cirrhosis.	Waxy.	Capsule thickened.	Waxy.	Tubercle.	Sanguineous apoplexy.
Inflammatory form (or nephritis)...	28	67.7	57.0	17.7	39.2	64.0	21.2	7.0	7.0	0	17.7	3.5	3.5	3.5	25.0	0	0	14.0	0	0	0	3.5	7.0
Waxy or amyloid (or lardaceous).	50	6.0	12.0	8.0	4.0	20.0	4.0	48.0	8.0	0	6.0	4.0	6.0	6.0	6.0	32.0	46.0	0	74.0	0	58.0	18.0	2.0
Contracting (or granular)......	13	23.0	53.7	7.5	46.2	53.7	7.5	23.0	7.5	0	15.2	0	0	0	15.2	0	0	15.2	0	38.2	0	15.2	15.2

CHAPTER XVIII.

ALCOHOL AS A CAUSE OF RENAL DISEASE.

In estimating the effect of spirituous liquors upon the kidneys, it is necessary to exercise considerable caution. Any agent which is thought to be powerful for evil is certain to be credited with mischief which it has had no share in producing.

The use of alcoholic drinks in some shape is almost universal. Among hospital patients in England there are but few male adults who cannot be convicted of a somewhat liberal use of beer or gin, while in Scotland whiskey-drinkers are relatively as numerous. There is probably no disease which is common in London or Edinburgh of which a majority of the men who suffer from it could not be convicted of intemperance in the article of alcoholic liquor. But to suppose that every disease which affects a person of such habits results from the action of the liquor is equivalent to believing that drunkenness confers a protection from all diseases excepting such as are consequent upon itself.

Among those who have become the subjects of renal disease we shall, unless alcohol act as a preventive, necessarily find a large proportion who have been somewhat liberal in the use of this stimulant. The statement of Dr., now worthily Sir Robert, Christison, that three-fourths, or even four-fifths, of the cases of granular degeneration of the kidneys which occur in Edinburgh are referable to a greater or less degree of intemperance, may be interpreted to mean that the majority of Scotchmen drink whiskey to what Dr. Christison considers an intemperate extent.[1]

In order to obtain evidence relating to the action of alcohol upon the kidneys, I have examined into the morbid appearances found in the bodies of persons who have died of delirium tremens, presuming upon such an end as evidence of inebriate habits; and also into those of persons employed in the liquor trades, potmen, draymen, cellarmen, and the like, who have been able to obtain drink without paying for it save pathologically, an opportunity of which the average Englishman seldom fails to take advantage; and finally I have collected such observations as appear to be trustworthy relating to the *post-mortem* condition of individuals who, having been notorious drunkards, have met with violent death.

From the records of St. George's Hospital from the year 1841 to the year 1871, I have been able to obtain the details of fifty-eight examinations, all of adult males, who had died in the medical or surgical wards of delirium tremens. From the same source I have taken for comparison the same number of the examinations of men who had met with accidental death, excluding such as had been notoriously drunken. It may be fairly presumed that the victims of delirium tremens had been on the whole more addicted to drinking than those who died of simple accident.

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[1] Christison on Granular Degeneration of the Kidneys, p. 110. In the term granular degeneration this author includes several varieties of renal disease.

Kidneys after death with Delirium Tremens, and from Accident without known Intemperance. All of adult males.

Condition of kidneys.	Delirium tremens, 58 cases.	Accident, 58 cases.
Natural	28	34
Congested	15	7
Slight or uncertain change in cortex	5	1
Large, smooth, mottled	3	1
Granular surfaces	6	8
Cysts without other change	1	7

Average age of delirium tremens patients, 38 years.
" accident " 41 "

Assuming, as we safely may, that most of the victims of delirium tremens have been habituated to alcoholic excess, we find in this fraternity of drunkards what to many persons may be a surprisingly small increase of renal disease. It must be observed, however, that the cases of accidental death are taken without exclusion as to trade, so that a certain proportion of lead-workers, of whose calling granular degeneration is a necessary result, have been admitted.

The slight increase of renal disease with delirium tremens is due to congestion and tubal disturbance; it is to be noted, however, as somewhat qualifying the conclusion to which the scarcity of granular degeneration in this series points, that the subjects of delirium tremens average three years younger than those of accident.

In the next place I reproduce, on opposite page, from the Medico-Chirurgical Transactions,[1] a statement compiled from the same sources, displaying the renal pathology of the brewery and gin-shop as contrasted with that of the non-alcoholic pursuits of the working class. The condition of the kidneys as observed after death is given in 149 dispensers of liquor, three of whom were barmaids, the rest were chiefly potmen, waiters, cellarmen, draymen, and publicans. For comparison is annexed a similar abstract from the same books, referring to the same number of grown persons of each sex taken (except that workers in lead were excluded as tending to obscure the result) without selection, save that they were all commercially unconcerned with liquor and none ostensibly inebriate.

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[1] The paper referred to is in the Medico-Chirurgical Transactions for the year 1873.

Post-mortem Condition of Kidneys in 149 *Persons of each class.*

	Engaged in the liquor trade.	Not engaged in the liquor trade.
NATURAL	49	48
HYPERÆMIA:		
Congested	18	18
Congested and enlarged	10	5
	28	23
TUBAL CHANGES:		
Coarse	3	3
Coarse and enlarged	4	1
Slight or uncertain change in cortex	2	4
Large, smooth, mottled [1]	5	9
Mottled, normal size, gray deposit in cones.	0	1
Smooth, pale, yellow, or gray, cortex shrunk	1	1
	15	19
FATTY CHANGE:		
Fatty or flabby	2	1
Fatty or flabby and enlarged	3	3
	5	4
LARDACEOUS	3	6
INTERTUBAL, FIBROID INCREASE:		
Cysts without other change	1	2
Cysts and depressions	3	1
Cysts and tubercles	0	1
Slightly granular	10	11
Highly granular	18	15
Granular + pyelitis	1	0
Granular + tubercles	1	0
Granular + conversion of other kidney into a cretaceous mass	0	1
Granular + stone	1	0
	35	31
Pyelitis	0	2
Abscesses in kidney associated with pyelitis.	1	6
Abscess from pyæmia	1	1
Abscess of uncertain nature	1	0
Tubercular disease	8	4
One converted into cretaceous mass, other natural	0	1
Cancer	1	1
Fibrinous blocks, without other change	0	2
Stone	2	1

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[1] It is possible that some of the kidneys thus described in the earlier records were lardaceous, in which case the preponderance of lardaceous disease in the non-alcoholic series would probably be greater than is represented.

Symptoms referable to Albuminuria in the preceding cases.[1]

	Engaged in the liquor trade.	Not engaged in the liquor trade.
Number of cases in which renal symptoms of any kind were prominent...................	20	26
Renal œdema occurred in......................	14	18
Internal dropsy, apparently renal, occurred in.	4	6
Albuminuria found in........................	23	29
Cerebral uræmia (coma, convulsions, etc.).....	5	3
Death from renal disease (directly)[2]...........	10	14

The preceding details, gathered as they are from careful *post-mortem* work, afford a comparison of a very trustworthy kind. The classes, indeed, which are opposed, do not contrast the results of alcohol with those of water—an unattainable comparison—nor even those of habitual drunkenness and invariable sobriety; but they show the difference of more and less. And that he who has liquor always at hand and for nothing will drink more than his fellow who must fetch and pay, is as certain as the virtue of opportunity.

The table shows, first that, as regards changes of every kind, congestive, tubal, or interstitial, which affect the secreting structure, the sum total under the greater influence of alcohol amounts to 86, under the less to 83. In detail it is seen that under the greater alcoholic influence were 14 instances in which the kidneys were either enlarged and congested, or enlarged and coarse, against 6 in the contrasted series. The swollen, somewhat hyperæmic kidney of the beer-drinker is indeed no unfamiliar experience. In the same class also is a somewhat greater tendency to fattiness of the epithelium; such an infiltration is found under the same influence in many organs and tissues. Next there is a larger frequency with the more liquor of interstitial fibrosis in the proportion as evidenced by granulation and cysts of 35 to 31; or as witnessed by granulation only of 31 to 27. To this extent we have evidence that alcohol affects the kidneys both tubally and intertubally; and to adjust the balance correctly we may even add a little to the alcoholic tale, on the score that the persons in this series who died on an average at 36 had less time to unfold their inherent proclivities than those with whom they are compared who reached the average age of 40. But with all allowance the increase is slight as compared with the increase under the same influence of disease in other situations. Cirrhosis of the liver occurred in 22 of the alcoholic to 8 of the opposed series; tubercle without distinction of seat with a scarcely smaller preponderance on the same side.

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[1] The table comprises all the renal symptoms observed in the cases of the two series, excepting those referred to pyelitis or renal abscess, tubercle, or other morbid growths, or calculus. These are excluded for the sake of placing the results of albuminuria in a distinct form.

[2] Sanguineous apoplexy is not included here.

As bearing upon the development of some kinds of renal disease, especially of such as are chronic, latent, and interstitial under an alcoholic vocation, a marked increase is to be noted under this influence of simple hypertrophy of the heart. Of this there were 15 instances in the alcoholic, 6 in the non-alcoholic series. Although it may be possible for this condition to be brought about by causes other than renal—changes in the blood and its channels—yet the association of simple ventricular hypertrophy with renal disease is so frequent that it is not possible to doubt that this proportion of cardiac change indicates a considerable, though perhaps not a proportionate, increase of renal fibrosis.

Some of the morbid states of the kidney appear to be nearly, and some absolutely, unconnected with alcoholic influence. The large white kidney of nephritis though, as I believe, exceptionally traceable to drink, is so much more often due to scarlatina and cold that the first mentioned agency shows no result in the table. And with regard to lardaceous disease, not only is this disorder less frequent under alcoholic pursuits than with others which carry a greater liability to the injuries upon which this change often ensues, but there is no reason on any ground to believe that the influence of alcohol is ever directly concerned in its production.

Finally, I may refer to a class of cases which, however, for the want of a standard of comparison, are less explicit in their teaching than what have gone before, and appeal to the valuable papers by Dr. Ogston,[1] who has recorded with minuteness the *post-mortem* appearances in the bodies of 117 persons of both sexes, who had been "by habit and repute" drunkards, and who met a sudden death by accident or suicide. The kidneys were natural in 68 cases, diseased in 49. The alterations were as follows:

Kidneys simply enlarged	7
Congested	13
Fatty	9
Granular degeneration, including such as were described as shrunk and cysted	7
Cortex simply shrunk	9
Nature of change uncertain	3
Containing an abscess	1

In these cases the most frequent departure from the natural state is congestion. Fatty transformation, or, in other words, occupation of the tubes by fatty epithelium, was noted in 9 cases of the 117, while granular degeneration occurred in but 7, a proportion probably not larger than would have been found in the same number of persons of ordinary habits.

Another writer, Dr. Peter, of New York, who has examined the bodies of nearly seventy persons "who had died from the excessive use of ardent spirits," thus sums up the changes observed in the kidneys:

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[1] See paper by Dr. Ogston, of Aberdeen, Med. Chir. Review, vols. xiii. and xiv.

"The kidneys are generally somewhat enlarged, flabby, their cortical substance infiltrated in numerous small spots, with a whitish matter, either albuminous or fatty. Occasionally they are granular."[1]

From the pathological facts which have been brought forward it will now be possible to form an estimate as to effect of alcohol in causing renal disease, which shall be based simply upon observation.

Lardaceous change may be at once put aside as having no association with this cause of disease. Great alcoholic excess may produce acute renal inflammation and the large white kidney, but the disorder of this origin is exceedingly infrequent compared to the instances in which it is traced to other causes. With the kidney, as with other organs, the effect of alcohol is in the production of chronic, not acute, changes. Subinflammatory tubal changes occur, evinced by various degrees of congestion and enlargement of the gland, and as in other organs under the same influence the epithelium may become fatty. Besides these tubal, or chiefly tubal, changes a result in interstitial fibrosis is to be recognized. Other causes of the granular kidney are greatly more frequent than is this; other results of drinking are greatly more frequent than is the granular kidney; but, nevertheless, this type of renal disease or some degree of the fibrotic exaggeration which is its essential, is an appreciable result of alcoholic excess. This is more clearly shown in the comparison based upon vocation than in any other of the statements which have been brought forward; and in this it is to be remarked that the frequency of simple cardiac hypertrophy, which may be taken in ordinary circumstances as of renal origin, is enhanced out of proportion to obvious renal disease. Possibly in some instances the kidneys were fibrotic when they passed for healthy; and not improbably the influence of the poison, otherwise than renally, may have had something to do with determining this condition.

As bearing upon the infrequency of granulation of the kidney as compared with cirrhosis of the liver as a result of liquor, I may mention that in forty cases of cirrhosis of the liver, in which that change had occurred independently of disease of the heart, and was for the most part associated with spirit-drinking, the kidneys were found to be granular but in eight, this disorder being generally in a comparatively early stage. These proportions show the remote subservience which the kidneys acknowledge to the property of alcohol, in virtue of which it renders the liver cirrhose.

The different relations which the kidney and the liver hold toward the stomach may explain the inequality which exists. Spirit, or anything which is absorbed by the gastric blood-vessels, is carried directly to the liver by the portal vein. It is then mixed with the blood of the ascending cava, and conveyed to the lungs, and cannot reach the kidney or any part of the general circulation until it has been subjected to the action of both the liver and the lungs, and become incorporated with the general mass of circulating blood. It may, therefore, be believed that alcohol, however tending to produce increased growth of fibrous tissue in the parts which it reaches in a comparatively unmodified form, exerts a smaller influence of this kind upon the kidneys. When, indeed, a large quantity has been taken, the whole system may be saturated, and alcohol may be excreted with the urine. A case is even reported by Dr. Ogston in which the urine of a person who had died drunk was so much charged with this fluid as to give off vapor which caught fire over the flame of a lamp. From such facts, and from the known diuretic action of alcoholic liquors, there can be no doubt that the kidneys take a share in removing any superfluity from the system, and it has been shown to what extent they suffer in consequence, but they are not exposed to the immediate action of the spirit, as are the structures which intercept it on its road and take toll before it reaches the general circulation.

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[1] New York Journal of Medicine, November, 1844.

In connection with the association of renal granulation with alcohol, a word may be said with regard to the intervention of gout. Liquor may cause gout, and with the gout the kidneys may become granular, but gout from liquor is less often thus accompanied than gout from lead ; it is better to be rich than poor, to drink port wine than to solder pipes or use a house-painter's brush.

Thus, while we allow the full proportion of renal fibrosis, which together with or independently of gout is to be attributed to the chronic irritation of alcohol, it is as certain as *post-mortem* observation can make it, that the liver, not for the present to touch upon morbid changes in the lungs, is more often affected by the same influence ; and as regards tubal and diffuse inflammation it is evident that their more acute kinds, though occasionally due to alcoholic excess, are so much more often produced by other causes, that this one is in this respect almost insignificant in its numerical results. Alcohol attacks other structures in preference to the kidneys. As causes of renal disease there are other agents, the more obvious and immediate have been previously considered, which are greatly more mischievous ; and there is a great atmospheric power, which overshadows all the other circumstances which tend to produce renal inflammation or granular change.

In the former edition of this work I supplemented the pathological evidence by some details gathered from the reports of the Registrar-General, but later research into these sources of information has brought out so much discrepant testimony that I am compelled to think with Dr. Roberts that, in the present state of nomenclature and registration, little trustworthy information as to the relations of alcohol and disease is to be obtained from these sources of information.

I append a table which was designed to show the prevalence of diseases of the kidney and liver under different degrees of intemperance and various habits of life; the prevalence of each cause of death is estimated in relation to the population as a sounder standard than the mortality; while the numbers are such as to afford large bases for inference. For the reasons I have stated I shall refrain to draw any conclusions on the present topic, though in some respects the table may not be without interest.[1]

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[1] 1861 was taken as the basis of the table as a year of census. The table, which has reference to the question of climate, as well as that of drink, is intended to show the relations of different modes of life in regard to shelter or exposure, as well as those of alcohol, to renal disease. With regard to the latter, the proportionate preponderance of alcoholism and of nephria or Bright's disease in London and the town districts of Scotland seems to point to a larger connection between the two causes of death than other information allows us to admit. But with regard to London in particular, and large towns in general, where there are hospitals, not only do these institutions attract the sick from rural districts, but in them disease is perhaps somewhat differently regarded and designated than in country places. The comparison, therefore, between town and country can hardly be exact. With regard to different beverages, we find less record of Bright's disease in the cider district than where beer or whiskey is the drink of the country. As touching differences in the circumstances of the laboring population, confining a large proportion of them to buildings or mines, or condemning as many to face all weathers, we find the advantage of shelter less apparent than might have been expected.

Proportion of deaths from Alcoholism, Diseases of Liver, of Kidney, Dropsy and Gout, compared to population, for the year 1861, *in several parts of Great Britain.*

		Total Population.	Deaths from alcoholism, 1 in	Deaths from liver disease, 1 in	Deaths from kidney disease, 1 in	Deaths from nephria, 1 in	Deaths from dropsy, 1 in	Deaths from ascites, 1 in	Deaths from gout, 1 in
Great Britain divided according to Beverage.	Beer District (comprising all England and Wales, except Devon and Hereford)..................	19,370,043	30,124	2,698	4,929	13,679	2,777	27,553	83,138
	Cider District (comprising Devon and Hereford)..	696,181	30,268	2.730	4.937	21,755	2,122	27,847	49,727
	Whiskey District (all Scotland).................	3,066,633	22,398	3,322	5,370	18,585	3,941	502,731	191,664
England divided chiefly according to Occupation.	London..	2,803,989	15,752	2,505	3,704	6,692	4,058	21,906	38,410
	Extra-metropolitan parts of Middlesex, Surrey, and Kent....................................	1,005,861	30,480	2,333	4,023	12,418	3,529	27,940	50,293
	Agricultural Counties (comprising Sussex, Hants, Berks, Herts, Bucks, Oxfordshire, Northampton, Huntingdon, Bedford, Cambridge, Essex, Suffolk, Norfolk, Wilts, Dorset, Somerset, Worcester, Rutland, Lincoln, Notts, and East Riding)..................................	5,482,142	43,857	2,635	4,649	17,570	2,515	29,410	62,297
	Agricultural Counties with admixture of mining, etc. (comprising Gloucester, Leicester, Cheshire, North Riding, Cumberland, Westmoreland, Shropshire, Monmouth, and North Wales).....	2,508,209	52,254	2,821	5,359	19,146	2,515	26,127	147,541
	Mining Counties (comprising Northumberland, Durham, Derby, and Cornwall)...............	1,543,872	36,758	2,752	5,937	17,745	2,477	44,110	385,968
	Manufacturing and Mining Counties (comprising Lancashire, West Riding, Staffordshire, Warwick, and South Wales)..................	6,025,970	26,318	2,779	5.867	15,611	2,768	26,546	194,380
Scotland Registration Districts.	Insular Districts............................	160,589	160,589	4,866	17,843	53,529	3,416	160,589	53.529
	Mainland—Rural Districts................... .	1,764,705	37.546	3.494	6.022	25,951	4.056	50,420	160,427
	Town Districts................................	1,141,339	13,587	2,935	4,227	12,141	3,855	45,653	570,669

CHAPTER XIX.

CLIMATE IN RELATION TO RENAL DISEASE.

Many diseases, like plants and animals, are distributed over the surface of the earth in obedience to laws which make living subservient to inanimate nature. If we know the range of temperature of a country, we can predict with some approach to accuracy the diseases which will be found among the inhabitants. In Arctic climates, catarrhal affections of the organs of respiration; in temperate countries, tubercular and renal disorders; within the tropics, dysentery, hepatitis, and malarious fevers prevail.

With regard to renal disease in particular, it will be necessary to follow its distribution somewhat in detail, so as to ascertain how far external circumstances are able to control its development. The recent spread of schemes of registration has given us a means of measuring the relative frequency of fatal diseases, in cities which present extreme differences of climate. The Army Medical Reports, under the superintendence of Dr. Graham Balfour, furnish the results of a series of experiments in geographical medicine such as could have been made in no age but the present, and by no nation but Great Britain. Our military posts are ranged like sentinels round the world; and extending from the Arctic circle to the Equator, and thence far toward the South Pole, they are exposed at the same time to every climate and every season. To their local influences we subject, with all the exactitude of scientific experiment, bodies of men of the same race, age, occupation, and habits, each with a staff of trained observers and an uniform system of recording the issue. Within a more narrow area we may appeal to the registrars' reports for England and Scotland.

To deal first with the more extended and probably the more exact source of knowledge, I may refer to a table published in the first edition of this work, which was compiled from the first six volumes of the Army Medical Reports, all which had as yet appeared, which showed albuminuria to be more frequent among the troops quartered in Great Britain and Australia than, to speak generally, in countries decidedly warmer than our own, India and the Cape, and the shores and islands of the Mediterranean. I have replaced the original table by one which gives later information, and comprises a greater number of particulars both as to disease and cli-

Proportion of Renal Disease among the British Troops in relation to the Temperature and Moisture of the Air in the various Military Stations of Great Britain; showing, unless otherwise stated, the yearly averages of Disease and Climate for Eight years, from 1865 *to* 1872 *inclusive.*

Places of garrison, arranged according to frequency of admission for Bright's disease.	Temperature, etc., at	Number of troops.	Number of years for which average calculated.	Bright's disease or nephria.		Nephritis.		Bright's disease and nephritis together.	Temperature of the air.			Hygrometer.				Syphilis.
				Admissions to strength, 1 to —.	Deaths to strength, 1 to —.	Admissions to strength, 1 to —.	Deaths to strength, 1 to —.	Deaths to strength, 1 to —.	Mean temperature.	Mean monthly range.	Mean daily range.	Mean difference of wet and dry bulbs.	Mean weight of vapor in a cubic foot of air.	Mean additional weight required for saturation.	Mean degree of humidity.	Admissions to strength, 1 to —.
Ceylon	Columbo	802	8	320	3,208	3,208	—	3,208	82.5	28.2	20.2	6.4	8.8	3.6	71.3	11
Japan	—	716	7	1,003	2,507	1,253	—	2,507	—	—	—	—	—	—	—	3.3
Bengal	—	35,222	8	1,241	5,635	1,394	35,222	4,858	—	—	—	—	—	—	—	11
West Indies	Barbadoes	1,458	8	1,292	—	1,292	—	—	80.1	20.1	14.6	8.1	7.8	4.3	64.1	10
China	Hong Kong	792	8	1,759	—	1,173	7,036	7,036	74.2	28.7	13.3	6.3	6.7	2.9	69.9	10
Australia	—	1,083	5	1,805	5,416	2,708	—	5,416	—	—	—	—	—	—	—	13
Bombay	Bombay	11,061	8	2,011	9,832	1,701	17,697	6,320	79.5	17.5	10.0	6.6	8.1	3.5	69.6	11
United Kingdom	Aldershot	78,615	8	2,124	7,146	2,833	48,378	6,226	50.4	38.1	16.9	4.3	3.4	1.2	76.2	11
Dominion of Canada		8,775	8	2,193	10,029	2,925	23,401	7,020								13
including Newfoundland	St. John's	292	4	585	585	1,170	—	585	41.7	50.2	23.5	2.4	2.8	0.7	81.3	39
" Canada	Quebec	9,026	4	2,139	19,253	2,750	38,507	12,835	40.3	51.7	20.9	3.6	2.7	1.2	71.3	10
" Nova Scotia	Halifax	3,218	4	3,218	12,873	3,218	—	12,873	43.4	47.9	20.6	3.1	2.9	1.0	74.2	24
New Zealand	Auckland	3,687	5	2,304	18,436	4,609	18,436	9,218	59.4	36.6	20.0	4.5	4.6	1.6	75.2	80
Madras	—	10,974	8	2,438	14,632	1,513	21,948	8,779	—	—	—	—	—	—	—	9
Gibraltar	Gibraltar	4,512	8	2,578	9,024	3,008	36,096	7,219	63.8	29.9	15.3	5.0	5.2	1.7	73.9	13
Cape, with St. Helena	—	3,742	8	2,993	—	1,663	—	—								6
including St. Helena	—	389	4	1,558	—	1,558	—	—	—	—	—	—	—	—	—	8
" Cape of Good Hope	Graham's Town	4,009	4	1,781	—	2,291	—	—	68.2	56.5	36.5	7.0	4.4	2.5	64.0	5
Malta	Malta	5,060	8	3,177	14,826	2,962	14,826	7,413	67.0	23.6	9.6	6.3	5.3	2.6	67.2	48
Bermuda	Bermuda	1,569	8	4,184	12,552	2,092	12,552	6,276	70.5	29.0	15.1	7.1	6.3	2.9	68.6	31
Mauritius	—	1,061	8	8,488	—	4,244	—	—	—	—	—	—	—	—	—	15

A blank (—) in a death column indicates that no death has been recorded in the series of years comprised in the calculation.

mate. The present table (p. 281) commences where the other ended, and covers the years from 1865 to 1872 inclusive. I have subjoined a statement of the frequency of syphilis at each station, as necessary to be considered in relation to the prevalence of albuminuria, a proportion of which, and in some instances a large proportion, is dependent on lardaceous disease. The figures referring to syphilis are calculated from the sum of admissions for primary and secondary symptoms. The admissions and deaths for Bright's disease, or, as it is phrased in the earlier reports, nephria, are kept separate from those spoken of as nephritis, since the latter term appears to be sometimes applied to painful lumbar disorders, not necessarily albuminuric. The greater weight must therefore be given to the variations recorded under the heading "Bright's disease;" though since it is probable that, when nephritis is fatal, it is always of the same nature, the deaths enumerated under this denomination may be taken into consideration, together with those which are certainly connected with albuminuria. The figures, whether relating to disease or climate, represent yearly averages. The statements refer only to British as distinguished from native troops. The stations are arranged according to the average of admissions from "Bright's disease" in relation to the strength of the garrison, beginning with the most frequent.

The equatorial districts, Ceylon, Japan, the greater part of India, the West Indies, and China, all give a large portion of renal disease. Our knowledge of the disorders which the tropics entail upon Europeans enables us to say that this is mostly lardaceous; not only does this systemic disorder flourish under a tropical sky, but in the regions specified it is to be noted that syphilis, one of its great causes, is especially prevalent. It is to be observed that attacks of albuminuria are more frequent in Bengal than in the other Presidencies; a difference which may possibly be associated with the greater moisture of its climate. Approaching the regions of the lesser renal proclivity it is first to be noted that in British America, with its low average and great variability of temperature, the albuminuric disorders, notwithstanding that the proportion of syphilis is not such as to indicate much lardaceous disease, are nearly as frequent as in the United Kingdom; in Newfoundland, indeed, with its exceptional humidity, they would seem to be more so, so far as we can venture to draw conclusions from the small number of troops imprisoned on that island of mist. With the higher temperature of New Zealand renal disease becomes less frequent, and is least so upon the subtropical shores and islands of the Mediterranean, and the solitary outposts of Mauritius and Bermuda where a tropical or nearly tropical climate is tempered by a vast circumference of ocean. In the subtropical range, which is thus comparatively exempt from renal disease, lie the Cape and St. Helena, which are, on the whole, lightly affected, notwithstanding the prevalence of syphilis at those stations.

I will now pass to information gathered from other sources, generally, perhaps, less precise than what has been already advanced, but by no means destitute of worth.

The mortality from renal disease in several great cities—taking cities together so as to compare similar communities—is placed next.

The proportion of deaths from renal disease to the total number of deaths is stated at, in—

		Mean annual temperature.
Aberdeen	1 in 49	47.0
London	" 89	50.8
Edinburgh	" 95	47.2
Dundee	" 107	—
Melbourne	" 110	57.
Glasgow	" 142	46.9
Paris	" 266	52.4
Bombay	" 2,800	80.6
Genoa	0 in 4,303	61.0

This is taken from published registers of causes of death. As renal disease, have been included all the deaths which, according to the phraseology in use in each register, are assigned to Bright's disease, albuminuria, nephria, nephritis or kidney disease. The numbers concerning the towns of Great Britain relate to the year 1863; with regard to the foreign cities, the latest sources of information in each case have been used. The places are arranged according to their liability to renal disease. The comparatively cold cities of Great Britain and Australia come first, Aberdeen occupying a bad eminence in this respect. Paris, though not differing very much in temperature from the warmer of these, has a somewhat less proportion of renal disease. Genoa, with its almost tropical climate, has an exemption which, for reasons which have been already alluded to, does not extend to Bombay.

The next source of information has been the reports of the Registrar-General for England and Scotland, which allow of comparisons to which some consideration must be attached, though no systems of registration record absolute truth.

The proportion of deaths from albuminuria or kidney disease to the total number of deaths from specified causes in Great Britain, in the year 1863, is thus displayed:

In England, 1 death in 106 was from this cause.
" Scotland, " 109 "
" Wales, " 131 "

Of the several divisions of Scotland:

In the Mainland rural districts, 1 in 103 was from this cause.
" Town " " 112 "
" Insular " " 188 "

The same facts, referring to many of the counties in detail, selecting those to which observations on climate can be appended, are given in a tabular form. The numbers refer to the year 1863 only, excepting where, from the smallness of the numbers or some other source of error, it was thought advisable to take the mean of two consecutive years; and in the cases of Shetland, Orkney, Caithness, and Sutherland, where the numbers refer to the whole length of time for which the detailed reports have been published.

Table showing the Mortality from Renal Disease in several counties of England with the annual Mean Temperature and the monthly range of variation.

	Deaths from renal disease.	Annual mean temperature.	Mean monthly variation of temperature.
Berkshire	1 in 71	50.7	34.3
Middlesex	“ 73	50.3	36.5
Dorset	“ 75	49.8	33.7
Sussex	“ 77	51.2	37.5
Surrey	“ 78	—	—
Cambridgeshire	“ 84	49.8	35.2
Devon [1]	“ 84	51.3	33.4
Kent	“ 84	50.3	36.5
Norfolk [1]	“ 84	50.1	33.4
Somerset	“ 90	49.2	34.9
Oxfordshire	“ 94	51.1	33.9
Warwickshire	“ 96	—	—
Northamptonshire	“ 98	—	—
Hertfordshire [1]	“ 103	49.2	36.9
Westmoreland [2]	“ 115	—	—
Cornwall [1]	“ 119	51.5	30.6
Northumberland	“ 120	46.8	30.6
Bedfordshire [1]	“ 121	49.3	29.2
Lancashire [1]	“ 137	48.7	33.0
Cumberland	“ 152	47.8	31.5
Durham	“ 155	—	—

Corresponding Table for Scotland.

	Deaths from renal disease.	Annual mean temperature.	Mean monthly variation of temperature.
Aberdeen	1 in 53	47.0	30.7
Caithness [3]	“ 67	—	—
Edinburgh [1]	“ 82	47.7	31.8
Fife	“ 94	47.4	31.5
Perth [2]	“ 104	48.5	33.8
Forfar	“ 107	47.8	30.3
Sutherland [3]	“ 118	47.0	30.6
Inverness	“ 127	46.5	23.4
Lanark	“ 143	46.9	26.6
Orkney [3]	“ 148	45.8	22.7
Ayr	“ 163	48.9	23.1
Argyll	“ 198	47.3	23.0
Shetland [3]	“ 487	43.8	19.9

[1] Observations from two positions within the county. Mean given.

[2] Deaths calculated for two years. [3] Deaths calculated for nine years.

The observations as to temperature refer to the year 1863 only. Where possible the mean of two stations within the county has been given.

The counties are arranged according to the frequency of renal disease, compared with other causes of death. It appears that the county of Aberdeen is pre-eminent among counties, as the city among cities, while the Shetland Islands, with their almost arctic climate, enjoy an immunity from the disease which is not approached by any other part of the kingdom. Further, it may be observed in passing, that within the limits of each kingdom, particularly in Scotland, where the climatic differences are more marked, there is a striking general correspondence between the amount of renal disease and the changeability of the temperature.

The details which have been brought forward, together with others which will be adverted to in passing, may serve as a guide to the geographical distribution of renal disease.

It appears that though lardaceous disease is frequent between the tropics, yet that albuminuria as a primary disorder, connected, that is, with renal inflammation or granular change, belongs especially to the temperate zone and to its colder districts. It is discouraged by a degree of warmth long short of equatorial, while, more tolerant of cold than of heat, it lessens on nearing the Arctic circle. It chiefly abounds where the mean temperature is not far removed from 50°. It is common in Paris, in Germany, in the United States, and in Great Britain.

In the southern hemisphere, exposure to a similar climate is attended with the same results. In Melbourne, with a mean temperature of about 57°, the disease appears to be scarcely less prevalent than in London. This appears from the results of general registration, while the mortality from kidney disease in the Melbourne civil hospital—one death in thirteen—is much what occurs in similar institutions at home. Among the troops quartered in Australia this disease, as has been made sufficiently apparent, is at least as frequent as in the United Kingdom. In New Zealand, with a very similar mean temperature, the army returns show less of it, though some of the comparative diminution must be ascribed to the paucity of syphilis in that virtuous colony.

At about the temperature of New Zealand the frequency of renal disease begins to diminish, and on passing the mean of 60° displays an extraordinary diminution. Renal disease, putting aside that of lardaceous origin, is the compatriot of wheat and barley rather than of the vine and the olive. It abounds wherever the climate, however cold during the winter, is warm enough in the summer, as in Canada, to bring wheat to perfection; and becomes scarce where oranges and lemons grow, and where deciduous trees as generally characteristic of the scenery are replaced by palms and other tropical endogens. In other words, it prevails wherever the heat for a considerable portion of the year is what would ordinarily be called temperate, whether usually so as in Great Britain, or so for a large portion of the year as in British North America, where comparatively mild weather divides the year with continuous frost.

In whichever direction we leave the temperate range we find albuminuria less common. In our own country, the colder Hebrides are comparatively free, while the Shetland Islands, with their ungenial summers and uniformity of cold weather, are less affected than any other part of the kingdom.

Still further North, I have been able, through the courtesy of Dr. Hjaltelin, the chief physician of Iceland, to whom I am glad to acknowledge my obligation, to import some facts of modern pathology from that ancient seat of learning and civilization. In answer to an inquiry, Dr. Hjaltelin writes:

"According to all circumstances which I have been able to observe in Scandinavia, Germany, and your native country, renal diseases are no doubt far more common in those countries than in Iceland; this is, no doubt, contrary to what might be expected, for as most diseases resulting from cold ought to be far more common than in temperated localities, so we might expect to find the Morbus Brightii very frequently in Iceland. This is, as aforesaid, by no means the case."

From this we may infer that albuminuria is rare in Iceland. "The shuddering tenant of the frigid zone," among his other advantages may boast his freedom from renal disease. And in the uninhabited polar regions it has been already shown that renal anasarca is not one of the disorders to which travellers are liable.

Pursuing the inquiry in the contrary direction, and leaving temperate for warmer latitudes, we find that, excepting when it occurs in the lardaceous form, as the consequence of other complaints, the disease diminishes as the temperature increases.

On the Mediterranean coast albuminuria is extremely rare. Dr. Chambers[1] ascertained that at Genoa, where the mortality is registered much as in London, kidney disease does not appear as a cause of death, while the deaths ascribed to anasarca or general dropsy are in Genoa 1 in 239, in London 1 in 93. The same writer compared the admissions at the Milan Hospital with those at St. Mary's Hospital, London, with the object of ascertaining the kinds of disease prevalent in the neighborhood of each. At Milan, Bright's disease, or dropsy with diseased kidneys, occurred on an average once in 2,807 admissions; while at St. Mary's, the proportion was 1 in 31, the affection being nearly a hundred times more frequent in London than in Milan, for the same number of patients. It is probable that this difference is somewhat exaggerated. From the rarity of the disease in Italy, it isnecessarily but little understood, and probably escapes notice more often than in London. English physicians, however, who practise in Rome speak of albuminuria as almost unknown there, excepting in association with phthisis, the disease being probably of the lardaceous variety, and consequent upon the pulmonary disease.

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[1] Chambers on the Climate of Italy.

Referring to the army reports, we find that the troops quartered at Gibraltar, Malta, and formerly in the Ionian Islands, are less amenable to albuminuria than those in Great Britain, New Zealand, or Australia, though from their necessarily short stay the climate tells less upon them than upon the permanent residents.

The same exemption from renal disease is found at the Cape of Good Hope, where the mean temperature is almost exactly that of Malta.

More nearly approaching the Equator we find that with tropical heat renal disease increases. The East and West Indies in this respect stand much in the same position; renal disease is frequent in the garrisons of both, though there is evidence that in the East Indies at least such disorders are comparatively rare among the civil population. We have the testimony of Sir Ranald Martin[1] as to the rarity in India of diseases of the lungs, air-passages, and kidneys; and the results of registration as concerning the city of Bombay represent that renal disorders are exceedingly rare compared with other causes of death; about one-tenth as common as in Paris, not a twentieth so common as in London. But as in other tropical countries albuminuria in some shape is less infrequent than in the subtropical, owing to the circumstances, among which the prevalence of dysentery must have prominence, which, under extreme heat, favor the development of the lardaceous disease.

Dr. Morehead, who describes Bright's disease as common among the hospital-frequenting classes of Bombay, gives the details of a large number of cases of albuminuria which came under his observation in a large hospital in that city, the patients being chiefly natives. In reading these ample and explicit reports it is not possible to avoid the conclusion that the disease was almost invariably of the lardaceous type, and very often the result of dysentery. Diarrhœa was a symptom of common occurrence, while uræmic disturbance was generally absent. In a large number of cases which were subjected to post-mortem examination, no instance was found of simple hypertrophy of the left ventricle, a change exceedingly uncommon as a result of the lardaceous disease, though generally present with granular degeneration. We cannot but conclude that the latter condition, and probably also merely tubal inflammation, is of rare occurrence in the Indian peninsula.

Thus, then, it has been shown that albuminuria, so far as it is dependent upon disease primarily renal, is more frequent in countries which have a similar temperature to our own than in those which are either much warmer or much colder. In the subtropical range the disorder, be it of what sort it may, is infrequent; between the tropics the kinds other than the lardaceous are rare; these kinds thrive with cold to a certain point, but do not appear among arctic complaints.

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[1] Sir R. Martin on the Influence of Tropical Climates, p. 639.

The prevalence of the disorder in temperate climates may probably be explained by the axiom that the liability of an organ to disease, particularly to inflammatory disease, bears a general proportion to its functional activity. The respiratory organs are the more active in cold, the kidneys in temperate, the liver and bowels in hot, climates. It has been shown that the urea, the chlorides, and the other constituents of urine, decrease as the air rises in temperature above 49°.[1] On the other hand, it is believed—a belief which is consistent with much of our knowledge—that the urea lessens also with severe cold.[2] Dr. E. Smith has shown that even in England during an exceptionally cold year, less urea was secreted by himself during the winter than during the summer months.

Within the limits of temperature in which primary renal disease prevails there is reason to believe that it is promoted by frequent and abrupt changes. Where the range is high, as at the Cape of Good Hope, there may be much variability with little renal disease; but where the temperature, as in the United Kingdom, is habitually low enough to encourage disease of this nature there, so far as we can trust the results of registration, it is the more abundant the more changeable the climate. In Scotland especially, which without much difference in the habits of the people comprises within a small compass great varieties of climate, and therefore is well adapted to throw light upon an inquiry such as occupies our attention, the correspondence between the amount of renal disease and the variability of temperature is striking. This is well shown by comparing the east coast with the west. On the western coast, where there is scarcely heat enough in summer to ripen wheat, but where the winters are warm from the influence of the Gulf Stream, so that a very uniform temperature prevails through the year, renal disease is not one-half so frequent as on the eastern side of the kingdom, where the weather is both colder and hotter than on the Atlantic shore, and undergoes much larger and more frequent variations.

It will be observed in the table that the Eastern counties all come at the beginning of the list, as having a large proportion of renal disease, the Western at the end, as most free, while Sutherland, which has a shore on each sea, occupies an intermediate position. The Islands, which resemble the western coast in their uniformity of temperature, resemble it also in their freedom from renal disease. The places where the temperature varies least are Ayrshire, Argyllshire, and the Orkney and Shetland Islands. These are also most free from such disease, Shetland having the pre-eminence in both respects.

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[1] Parkes on the Urine, p. 95.

[2] Philosophical Transactions, 1861.

It appears then, that within the colder temperate range the amount of primary renal disease is regulated in part by the variability of the temperature.

The dependence of renal disturbance upon such fluctuations is in accordance with all our knowledge. The phenomena of "catching cold" result, not so much from continued low temperature as from abrupt transitions; and the association of the tubal disorder with the series of changes thus familiarly described, has been already shown.

These considerations may explain a circumstance which at first is a little surprising, namely, that marked as is the effect of climatic warmth in lessening some kinds of renal disease, the same result is not noticeable with regard to more partial or artificial temperature. Persons who toil in the heated atmosphere of mines, or are protected from weather in manufactories, do not appear, by the registrars' reports, to suffer the less on these accounts from renal disease. (See table, p. 297.) The proportion of deaths ascribed to Bright's disease, to kidney disease, or to dropsy is not markedly less in the mining and manufacturing counties than in the agricultural. Probably the explanation lies in the inconstancy of the protection.

We must also attribute some influence, though apparently only a minor one, to humidity of atmosphere as a condition which must necessarily interfere with cutaneous action. The subjects of chronic renal disease are, as a rule, sensitive to this condition; they shun damp and relaxing climates, and seek those which are dry and bracing; but the influence of climate is made up of so many items that it is difficult to assign their exact weight to the less important. A reference to the table will show that of the military stations, putting aside those within the tropics for reasons which have been sufficiently dwelt upon, the greatest freedom from renal disease is associated, as on the Mediterranean, at the Cape, and at Bermuda, with an atmosphere which, however much water it contains, has a large capacity for more; and together with this it may be taken for what it is worth—the small size of the garrison forbids a confident conclusion—that in Newfoundland, where the humidity is at its maximum, there also is the largest mortality from renal disease. It is probable, however, that humidity is of less consequence than temperature, as, to judge from the registers, the west coast and islands of Scotland are, notwithstanding the greater dampness of their atmosphere, less amenable to the disorders in question than the dryer and more variable eastern shore.

In the preceding account it has not been generally practicable to separate the several renal disorders from each other, but it is sufficiently evident that the variations which have been described depend, in temperate climates at least, upon the prevalence of the inflammatory affection, and of granular degeneration, more especially of the latter as the more common disease.

With regard to this disorder, so frequent and so little under the control of treatment simply medical, the influence of climate has a great practical importance. This can scarcely be said with regard to the other diseases which have been considered.

The inflammatory affection, indeed, may owe its origin to atmospheric influences, but is generally so rapid in its course and urgent in its symptoms that the subjects of it have no choice but to stay at home. The patient recovers or dies without any interval of health such as would allow of his removal to a foreign country.

The lardaceous change, not belonging especially to the kidneys, owes its origin to circumstances within the body rather than external to it; and so long as such sources are in operation it will number its victims under every sky.

With granular degeneration, however, the case is otherwise. It is a very chronic disease, and it appears to have a close dependence upon atmospheric influences. The structure of the organ is invaded by slow and often hesitating approaches, and we cannot but admit the importance of any measures which, by removing the tendency to the disease, may save as much of the gland as is yet intact. That the tendency to the disease varies in different countries has been abundantly shown. And I am now able to add the testimony of experience to what in the earlier edition of this work was little more than anticipation as to the salutary effect of removing a patient in whom the earlier symptoms of the complaint have been discerned to a locality where the genius of the place is opposed to its progress.

In directing the line of retreat it is to be borne in mind that renal patients are often remarkably susceptible to the depressing influence of damp and relaxing climates, and are readily exhilarated by one which is dry and bracing. What is described as a stimulating climate suits them. Those who cannot leave England are better, so far as I have seen, at Brighton or Folkestone than at Lyme Regis or Torquay; or to point to a place where during a considerable part of the year at least warmth and dryness are to be found together, Bournemouth may be suggested. The benefit, however, to be gained by removal from one part of England to another is not such as to make it generally worth while. A greater change gives better results. Beyond the limit of the four seas the distribution of renal disease indicates a subtropical rather than a tropical climate. The desiderata appear to be mainly an even temperature, with a mean between 60° and 70°, and a dry air. The choice may range over all the coasts and islands of the Mediterranean, Egypt, the Cape, and its vicinity. One of the nearer of the places thus indicated may serve as well as the more remote, and among the towns and villages of the Riviera, Mentone deserves especial mention as having a climate which is found to suit the subjects of albuminuria. A gentleman, who wintered abroad to the perfect restoration of his general health, and the reduction of the albumen to a trace, pronounced the Riviera cold and Egypt chilly, and never experienced a satisfactory warmth until, in its pursuit, he found himself at Bombay. But most persons may well be content with Mentone, Cairo, or the Cape. For those, however, to whom India, or one other of our equatorial possessions is placed by circumstances within easier reach than less distant resorts, the tropics, with all their dangers, may be preferred to our own chilly and changeable climate and the almost certain progression of the disease.

That as a rule in our own climate the granular kidney is inevitably progressive need not be reasserted. The remedies of art in such a case may be hopefully exchanged for those of nature ; and in the impotency of such medicine as is represented by the Pharmacopœia, we may yet be able to take advantage of those universal laws in virtue of which all life and growth are regulated by the physical conditions of the earth.

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